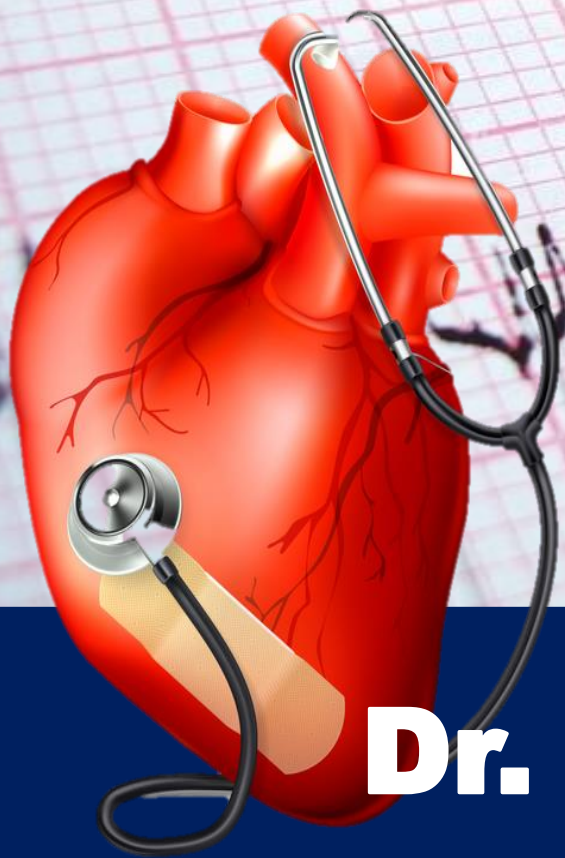




Dr. Tarek Abdelhamid

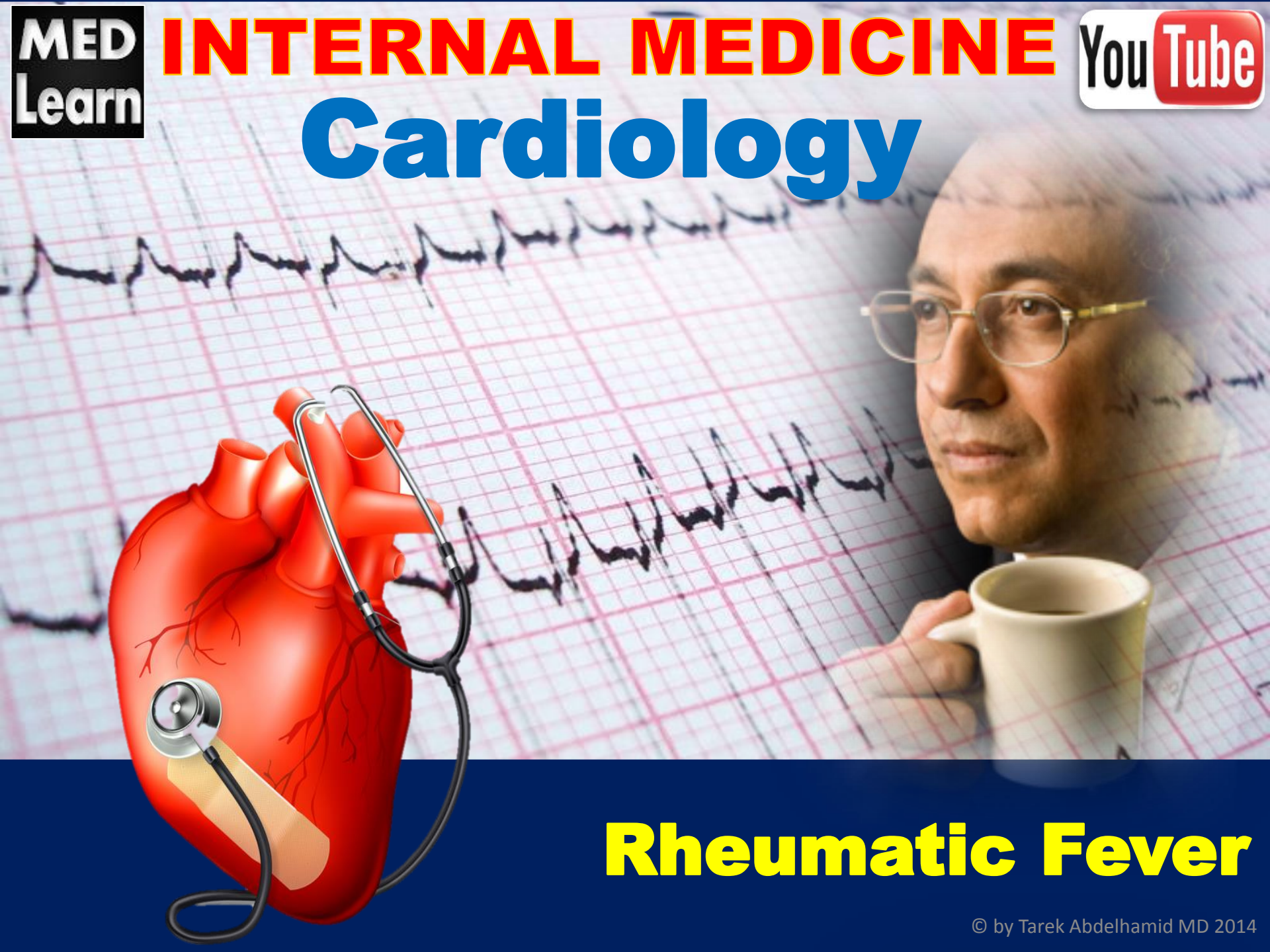
VISIT...

LANZAROTE
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Tarek Abdelhamid M.D.

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Cardiology

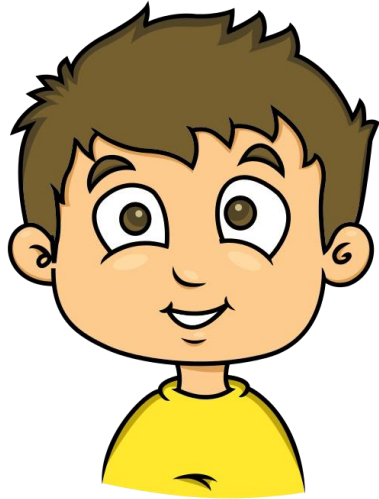


Rheumatic Fever

Definition

A systemic immune response that occurs as a reaction to infection by β Hemolytic strain of Streptococci.

Aetiology

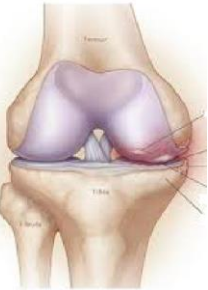


Recent infection by
group A β -
Hemolytic strain of
Streptococci e.g.
sore throat

This has to be
treated Early to
decrease the
incidence of
Rheumatic Fever.

Clinical Picture

Major Criteria



Arthritis

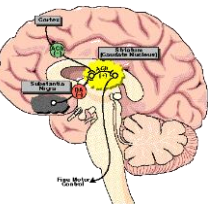
- 1- Big Joints
- 2- Fleeting
- 3- Respond to Salicylate
- 4- No consequences



Carditis

Pancarditis

- ① Myocarditis: Heart Failure
Arrhythmias
- ② Endocarditis: Valvular lesions
- ③ Pericarditis



Chorea

Abnormal movements of the hands
(female)



**Aschof
nodules**

Subcutaneous nodules:
Painless- behind the
Mastoid and Olecranon
process.



**Erythema
Marginatum**

Rapidly enlarging
macules on the
trunk

Clinical Picture

Fever

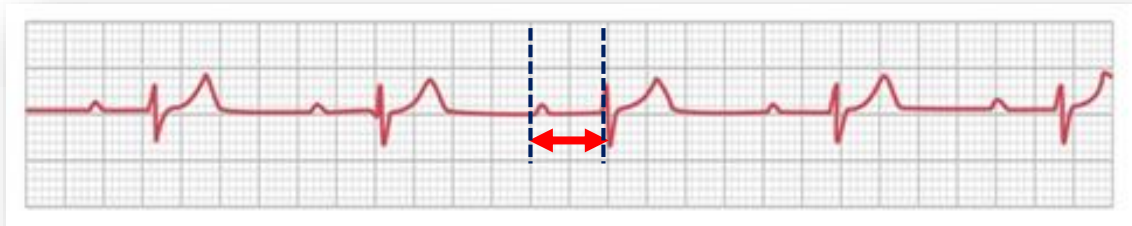
Pallor

Epistaxis

Arthralgia

+++ PR
Interval

ECG



↑ ESR

↑ CRP

↑ WBCs

Minor Criteria

Diagnosis

2 Major

1 Major + 2 Minor

Plus

**Any evidence
of recent
Streptococcal
Infection**

Notes



Note 1

MS

Oedema of
the Mitral
Valve



Diastolic Murmur

MDM + **↑**S1

MDM But **NO** **↑**S1
(Reversible: **Carey Coombs murmur**)

Note 2

MR

Fever



Systolic Murmur

Systolic Murmur with **↓**S1

HKC Systolic Murmur
But **NO** **↓**S1

Special Investigations

 **ASOT**

- 1- Anti- Hyaluronidase
- 2- Anti- NADase
- 3- Anti- Streptozyme test

 **ESR**
 **CRP**
 **WBCs**

Echo



Treatment

Prophylactic Treatment

Benzathine Penicillin G 1.2 million units IM every month until Age 35 Ys.

If the patient is hypersensitive to Penicilline use **Sulpha**.....(Repeated Blood Count and Urine Analysis)

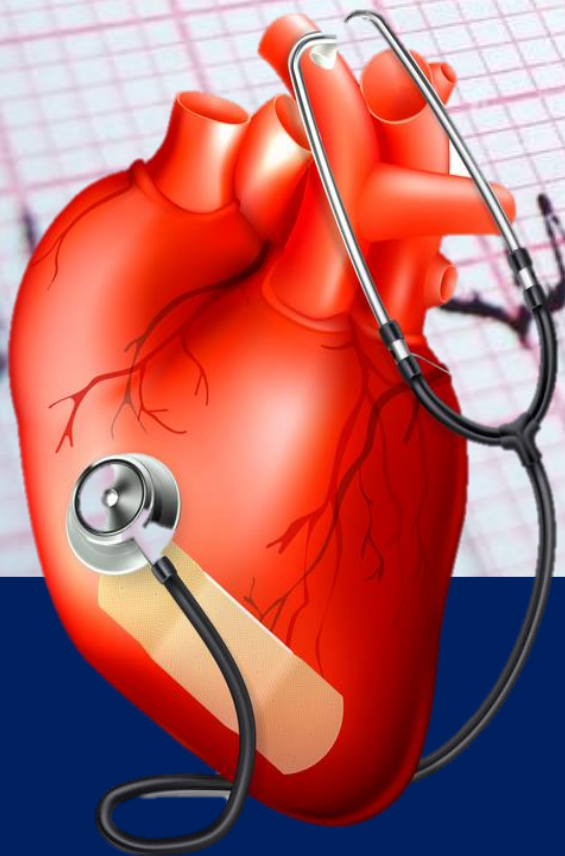
Active Treatment

Salicylate for the Arthritis: 10 mg/ day for 1 Week then 5 gm/ day for 1 Month (on 6 divided doses to avoid gastric irritation)

Corticosteroids for the Carditis: Prednisone 1mg/Kg BW for 3 Weeks

NO Na in Diet if the patient has Myocarditis

Cardiology



SBE

Definition

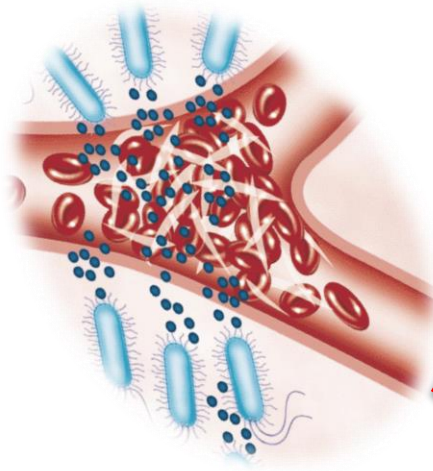
It is a proliferation of microorganisms on the endothelial surface of the heart.

Aetiology

The organisms that can cause SBE include:

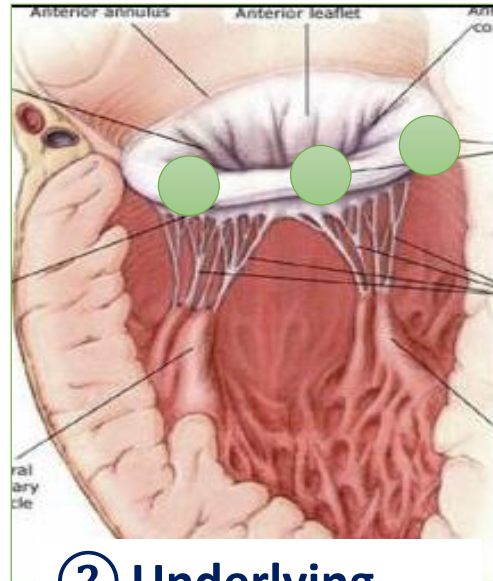
- ① **Streptococcal Viridians** (?! Commonest organism)
- ② Streptococcal faecalis
- ③ Staph aureus (the Most Virulent form)
- ④ Fungal infections (in IV drug addicts)
- ⑤ Gm -ve organisms (after Urinary Procedures)
- ⑥ Rickettsial infections e.g Q fever

Pathogenesis

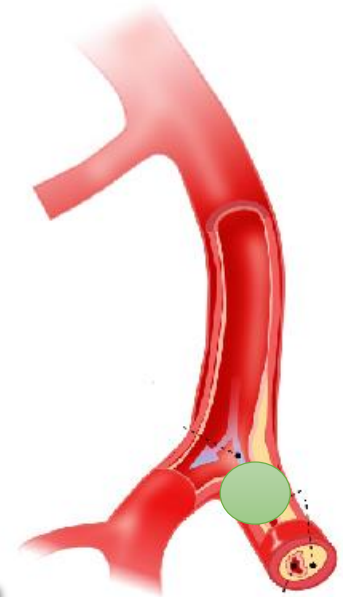


① Source of Bacteremia

③ Vegetations
(Damage the valve)

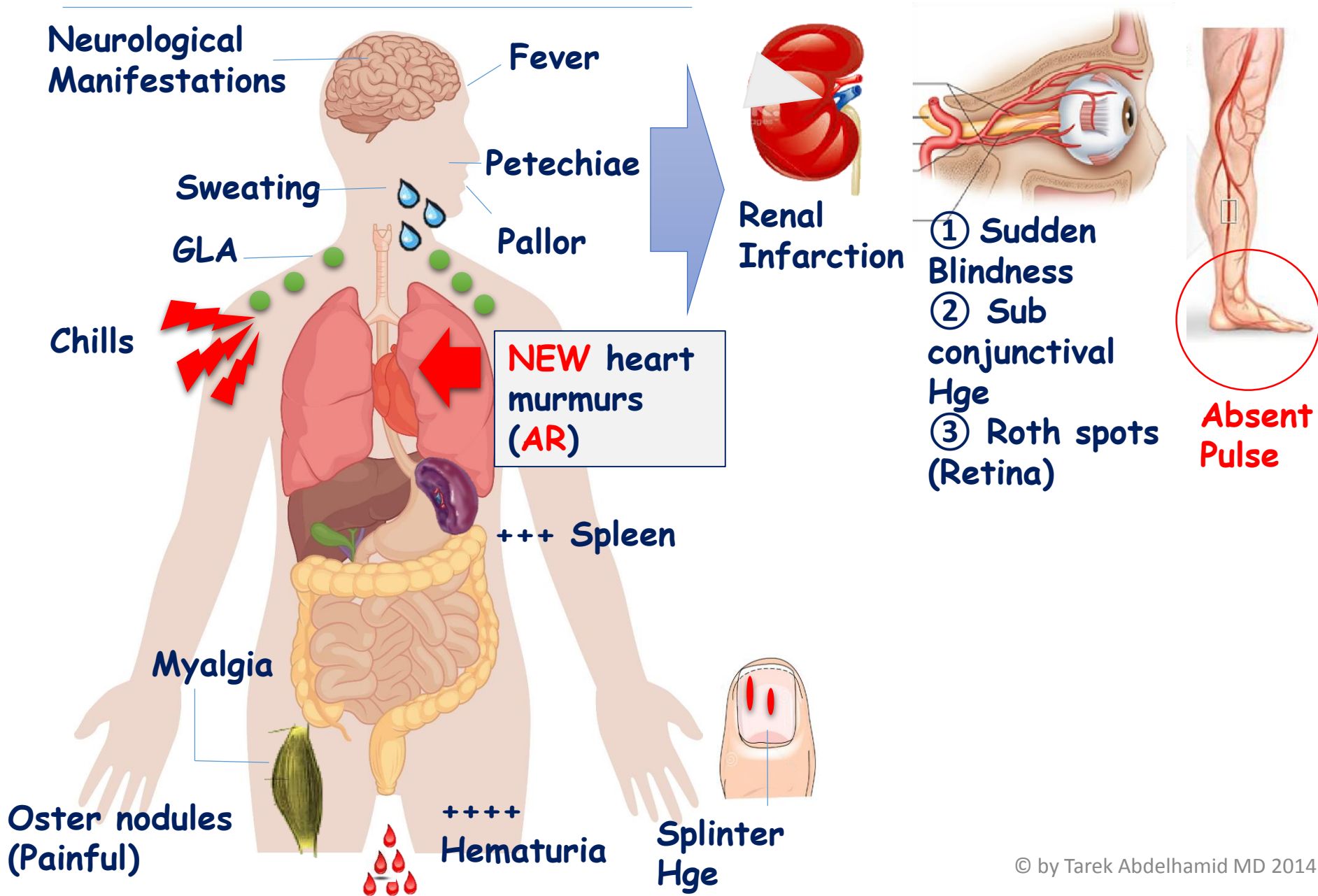


② Underlying Cardiac lesion



④ Embolism

Clinical Picture



Notes:

Note 1

If any patient with underlying cardiac lesion e.g. Valvular heart disease – congenital heart disease- AMI- developed **FUO plus Murmur of AR (EDM).....** You should suspect SBE.

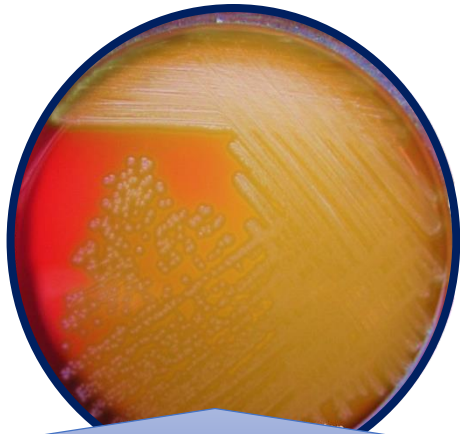
Note 2

Fungal Endocarditis

- ① May occur on T.V. especially in IV drug addicts
- ② Culture –ve endocarditis
- ③ Associated with Big Vegetations that may obstruct the T.V. (Stenosis)...this could be detected on Echo
- ④ this condition is suspected clinically if the patient's Heart Failure is Resistant to treatment.

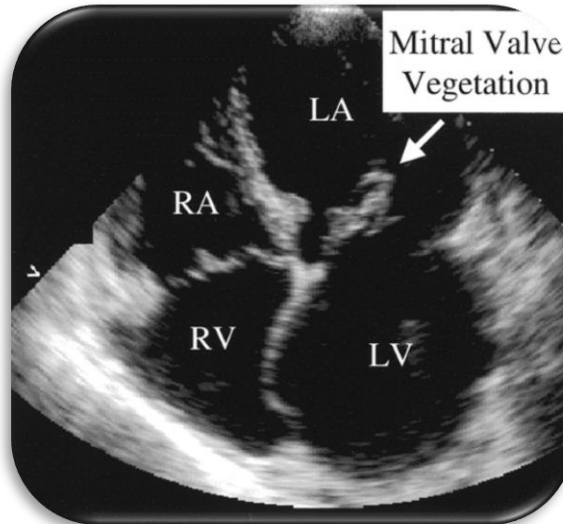
Special Investigations

+ve Blood Culture
(Most Important)



Positive blood
Culture in SBE

Echo: to detect the
Vegetations



**Immune
Complexes**

 **ESR**

**+ve
Rheumatoid
Factor**

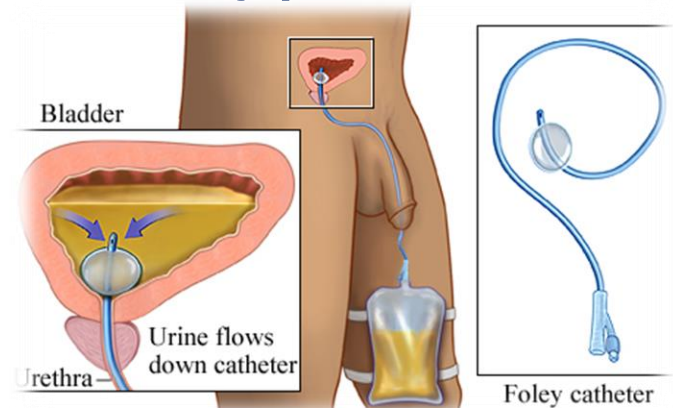
Treatment

Fungal Endocarditis

① Prophylactic Antibiotics should be given to patients who undergo tooth extraction or Urinary procedure



Amoxicillin 3 g Orally before dental procedure

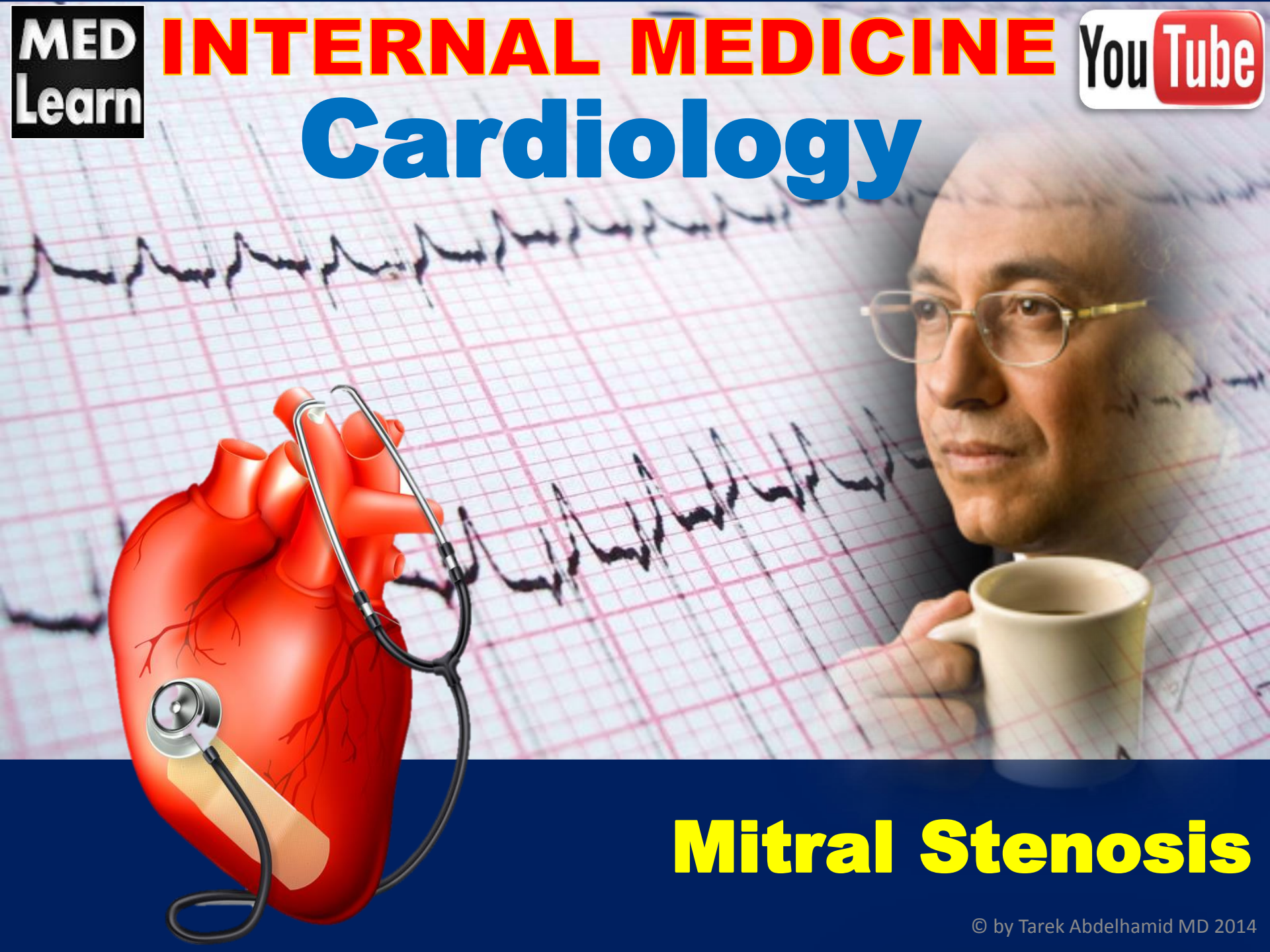


Ampicillin 1 g orally **plus** **Gentamycin** 80 mg IM before Urinary procedures.

② Active treatment should include antibiotics that covers Staph- Strept- Enterocoli e.g. (Nafcillin-Penicillin (or **Vancomycin**) plus Gentamycin.

③ Surgical Removal of the infected valve may be used if Medical treatment is not satisfactory.

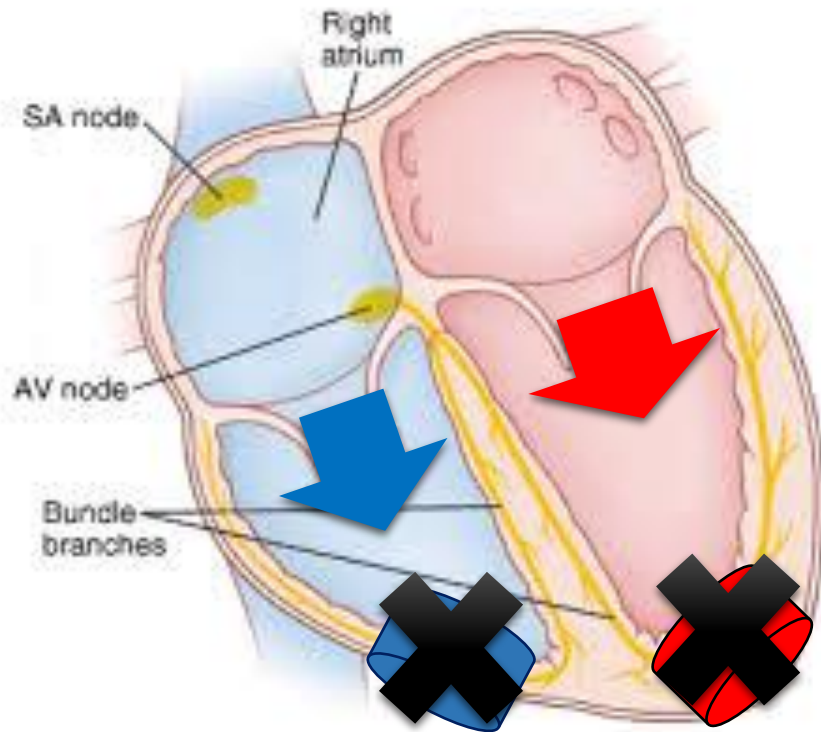
Cardiology



Mitral Stenosis

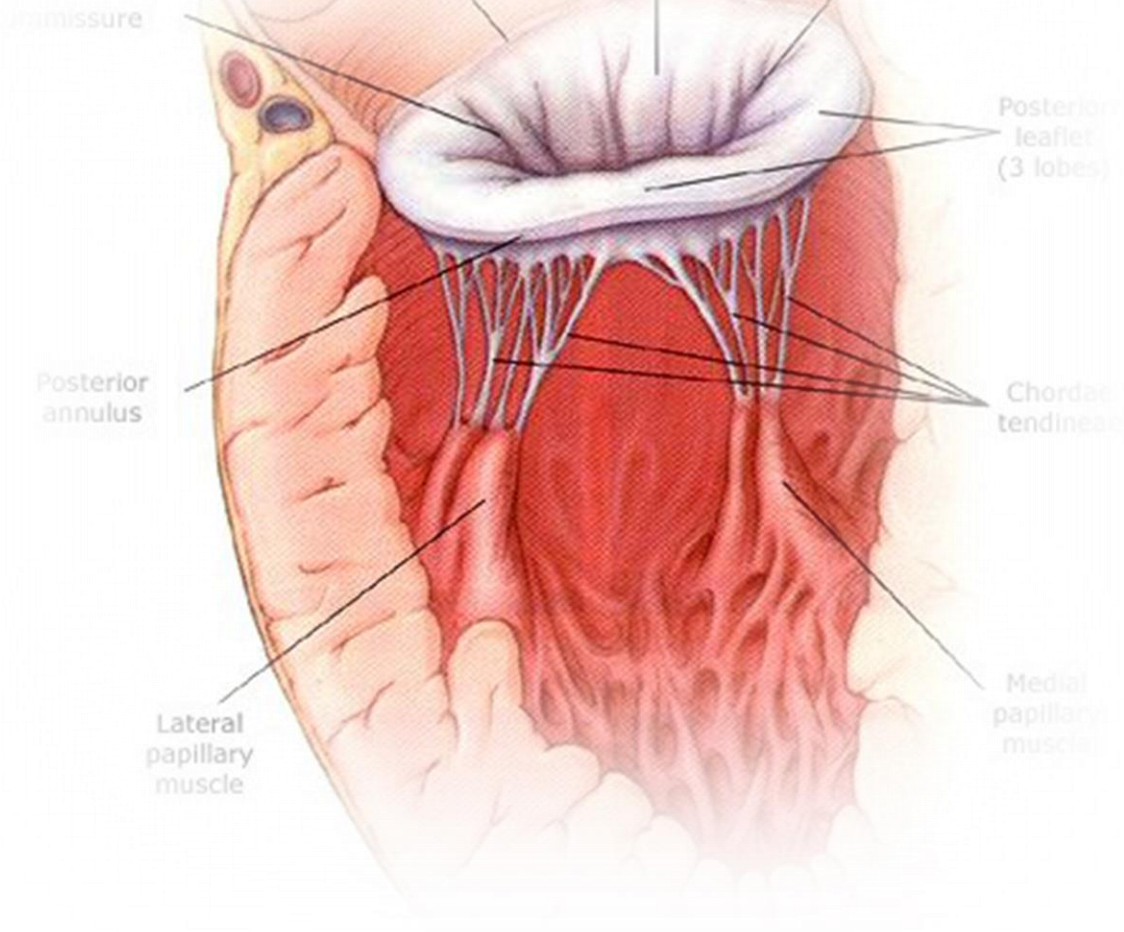
Definition

Inability of the Mitral Valve to open properly when it needs to be open (i.e. in Diastole)

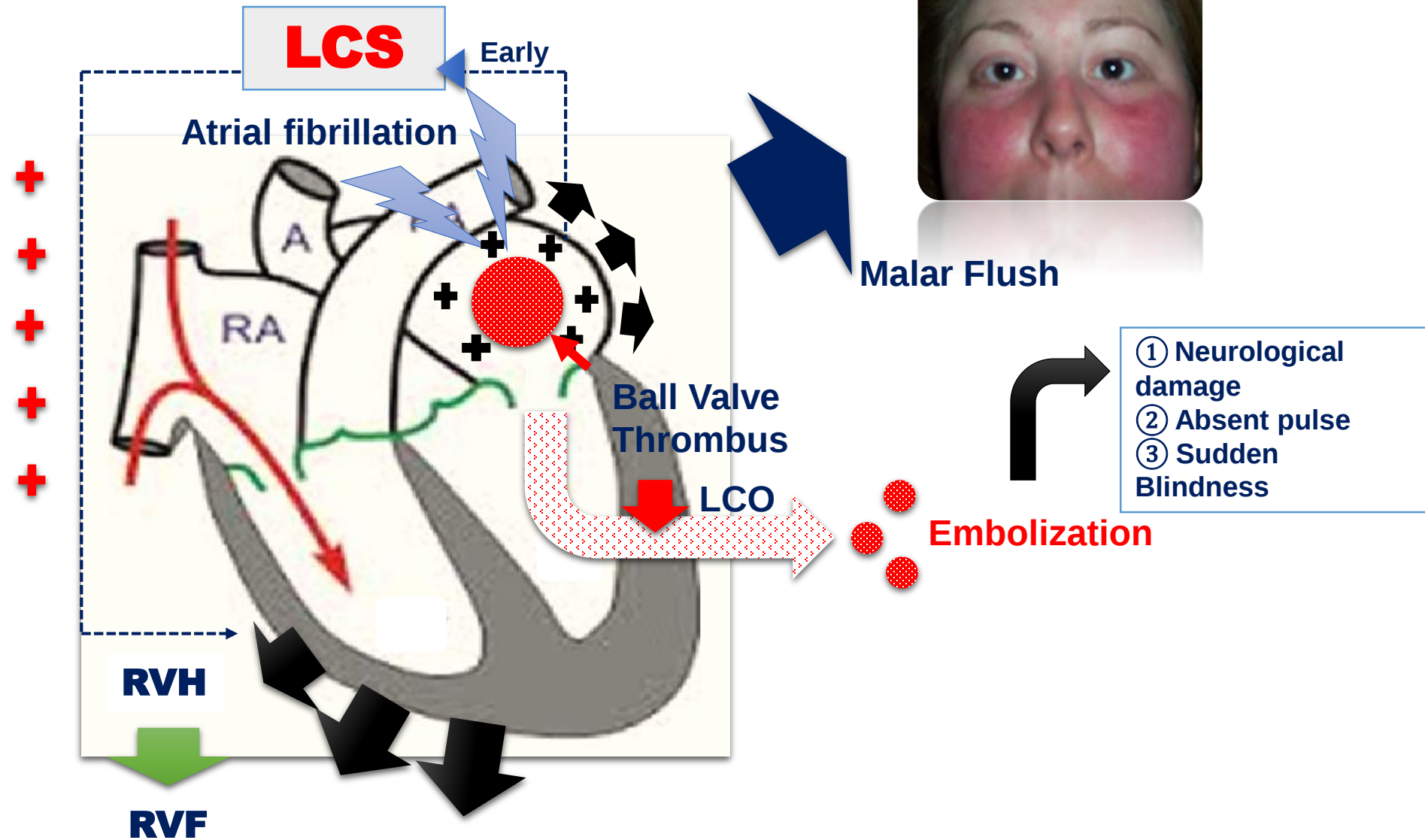


Aetiology

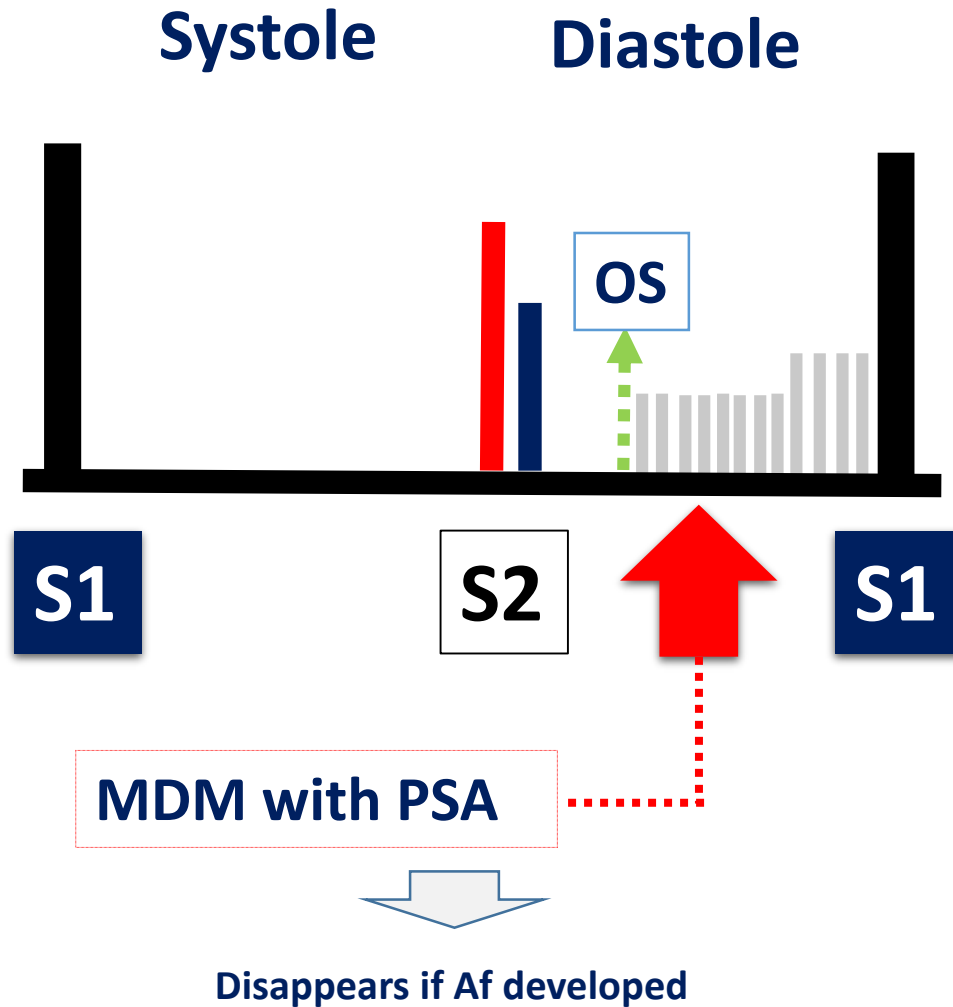
Rheumatic Heart Disease is the Most Common Cause



Clinical Picture



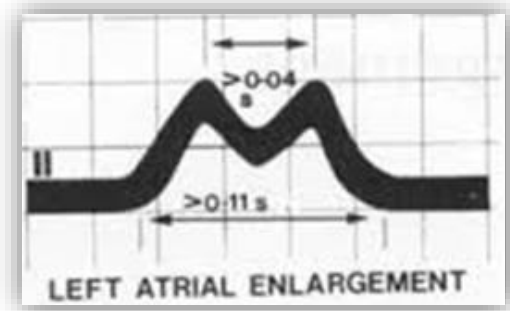
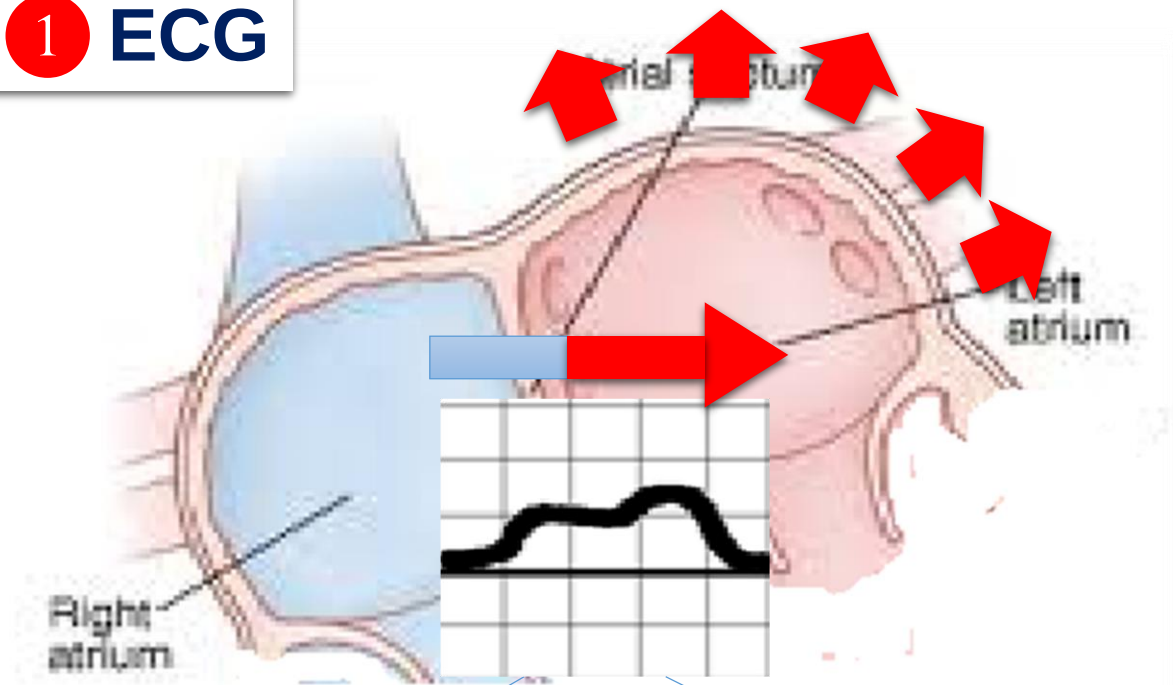
Auscultation



Special Investigations

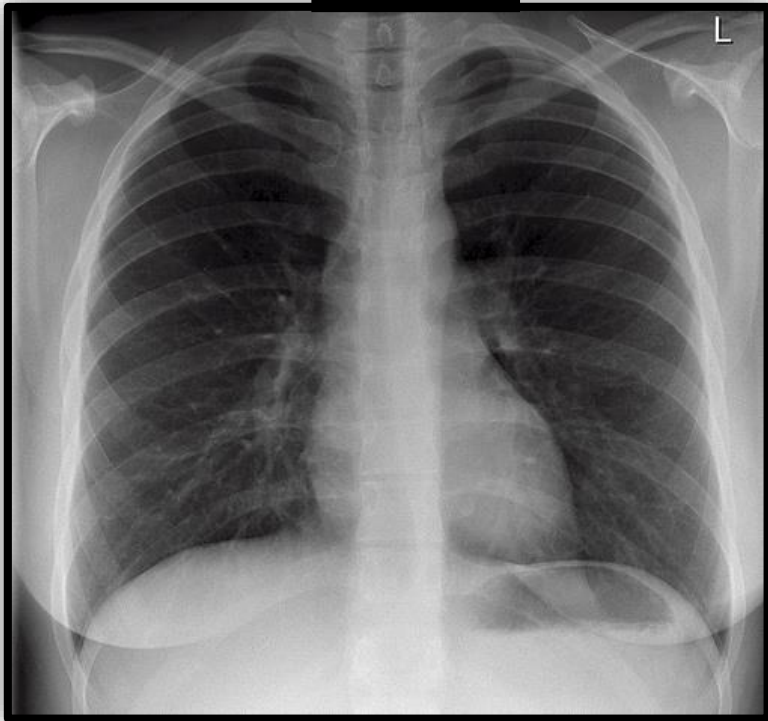
- 1 ECG: P-Mitrale
- 2 X-Ray: Butterfly Wing Appearance- LAH- RVH (Later)
- 3 Echo: Diagnostic

1 ECG

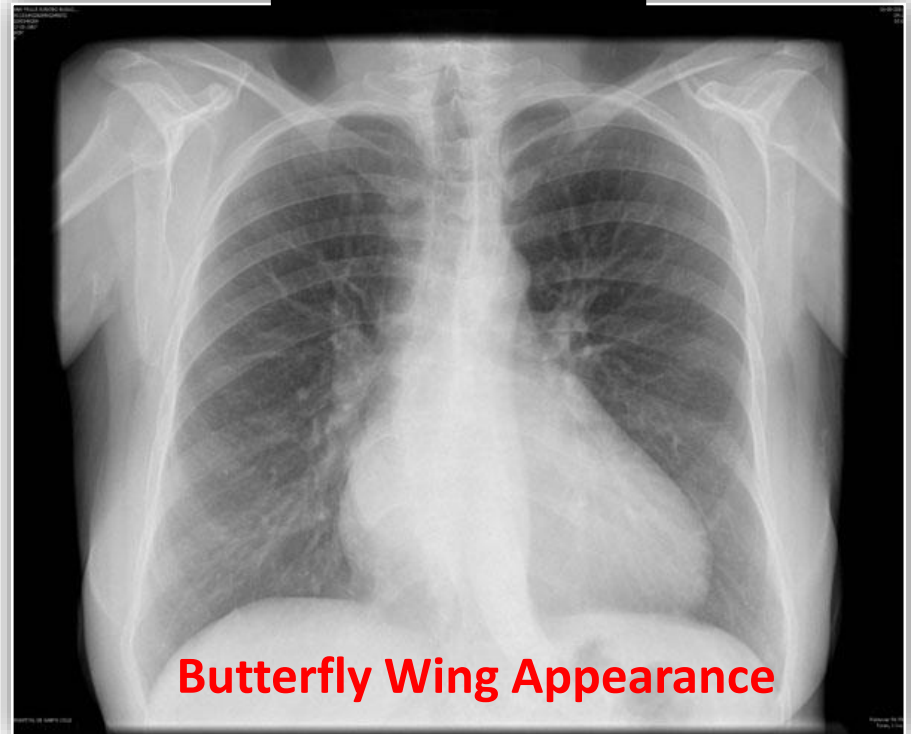


2 Chest X-Ray

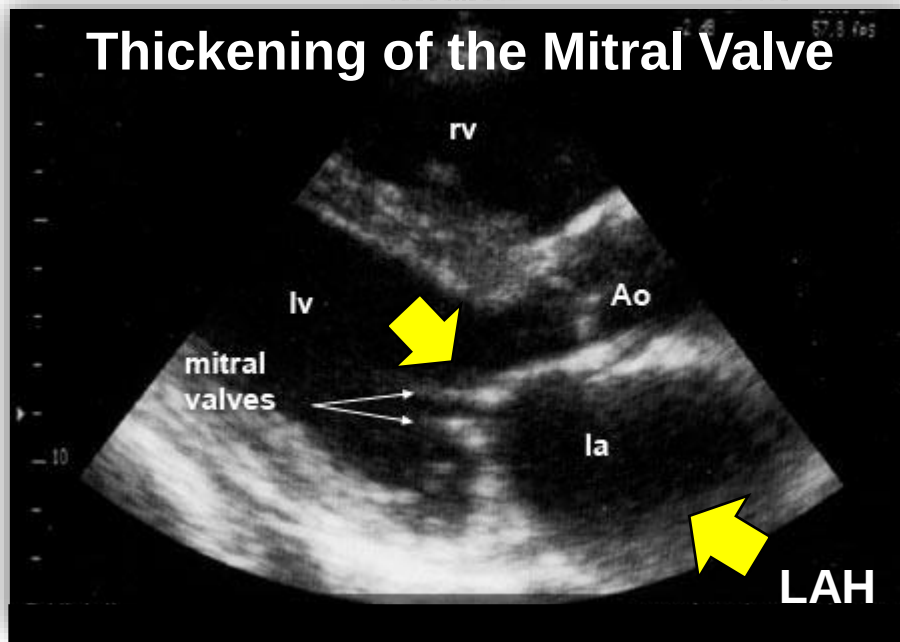
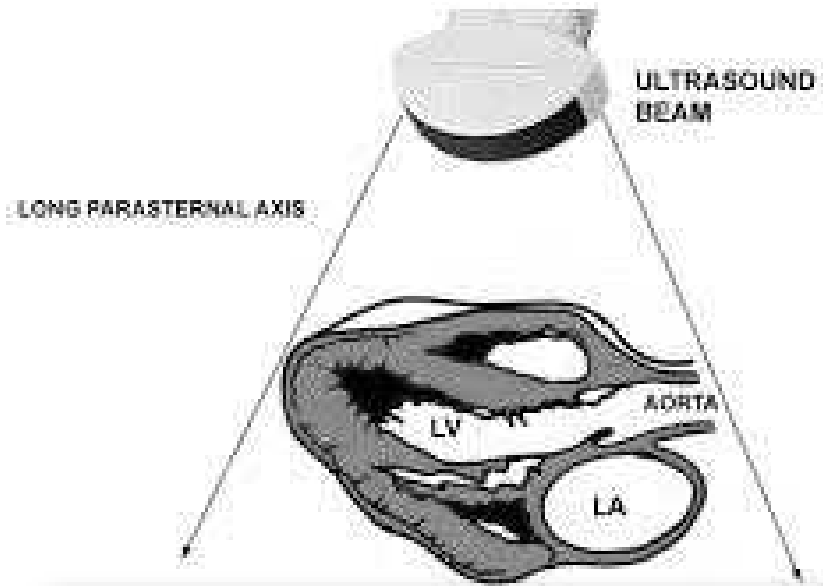
Normal



Lung Congestion

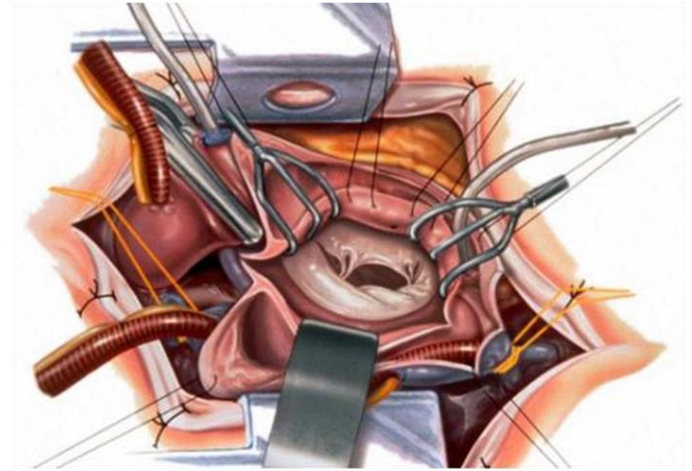


3 Echocardiography

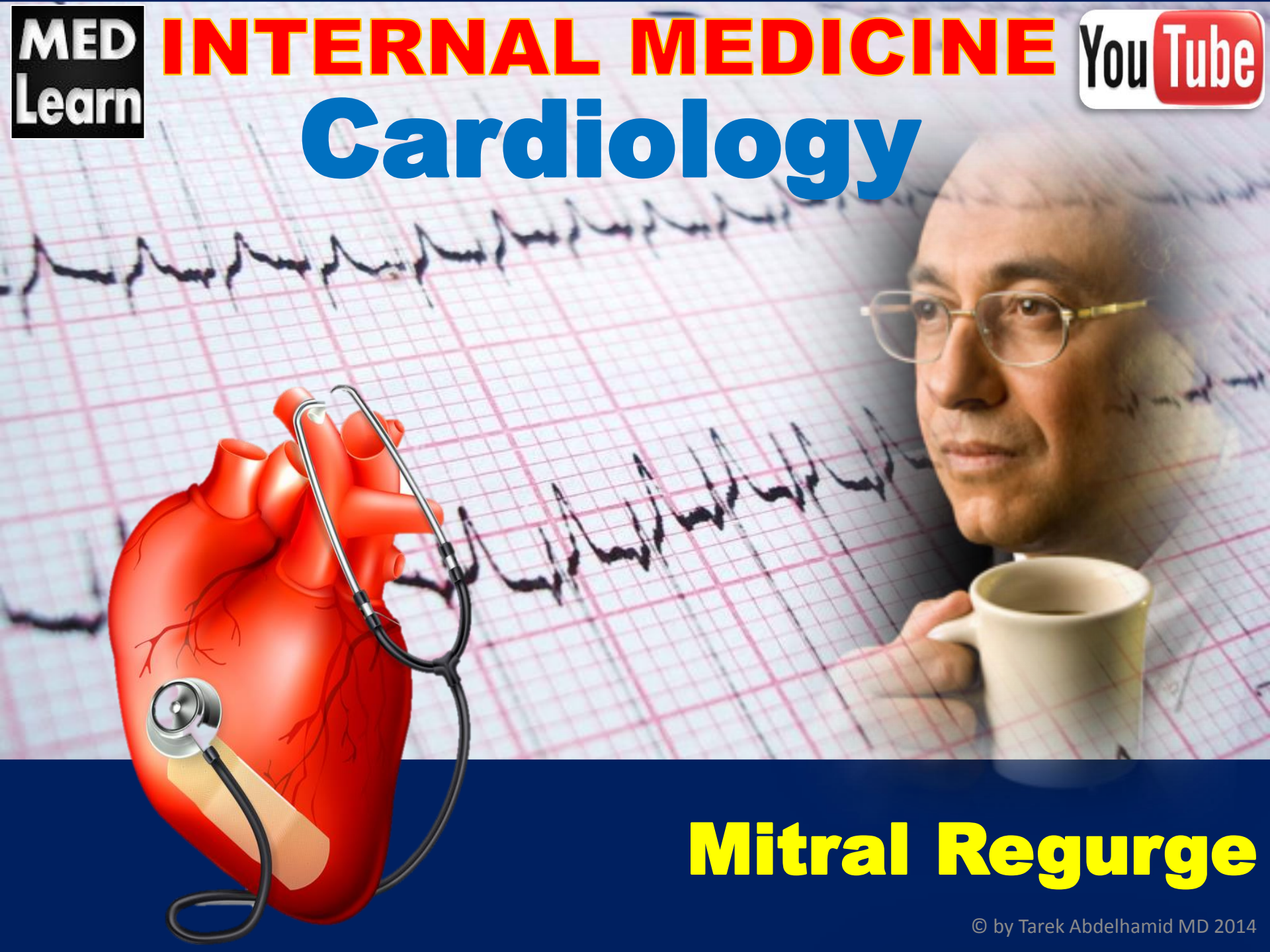


Treatment

- ① Prophylaxis against Rheumatic Fever and SBE
- ② Anticoagulation if Af or after embolization
- ③ Surgery (Valve replacement)



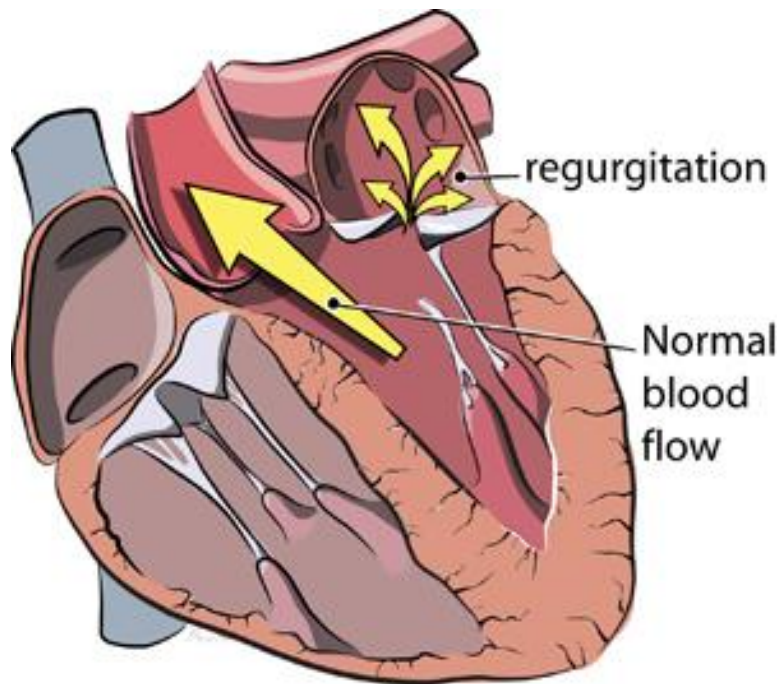
Cardiology



Mitral Regurge

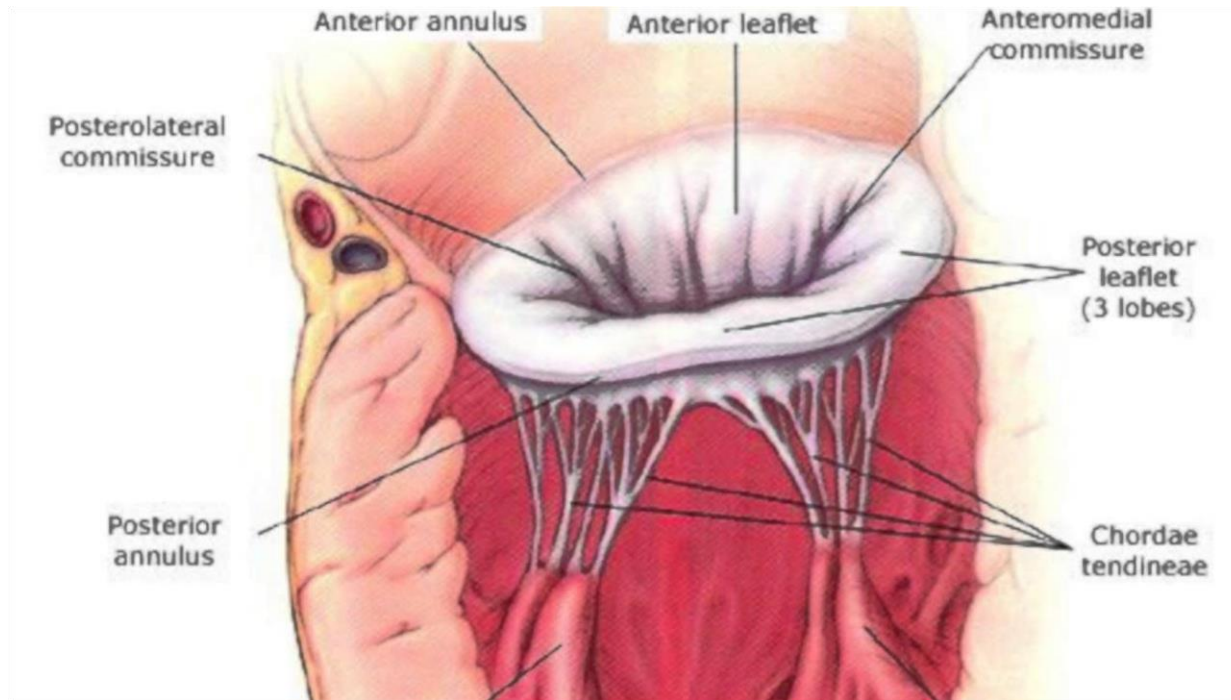
Definition

Inability of the Mitral Valve to close properly when it is needed to close (i.e. in Systole).

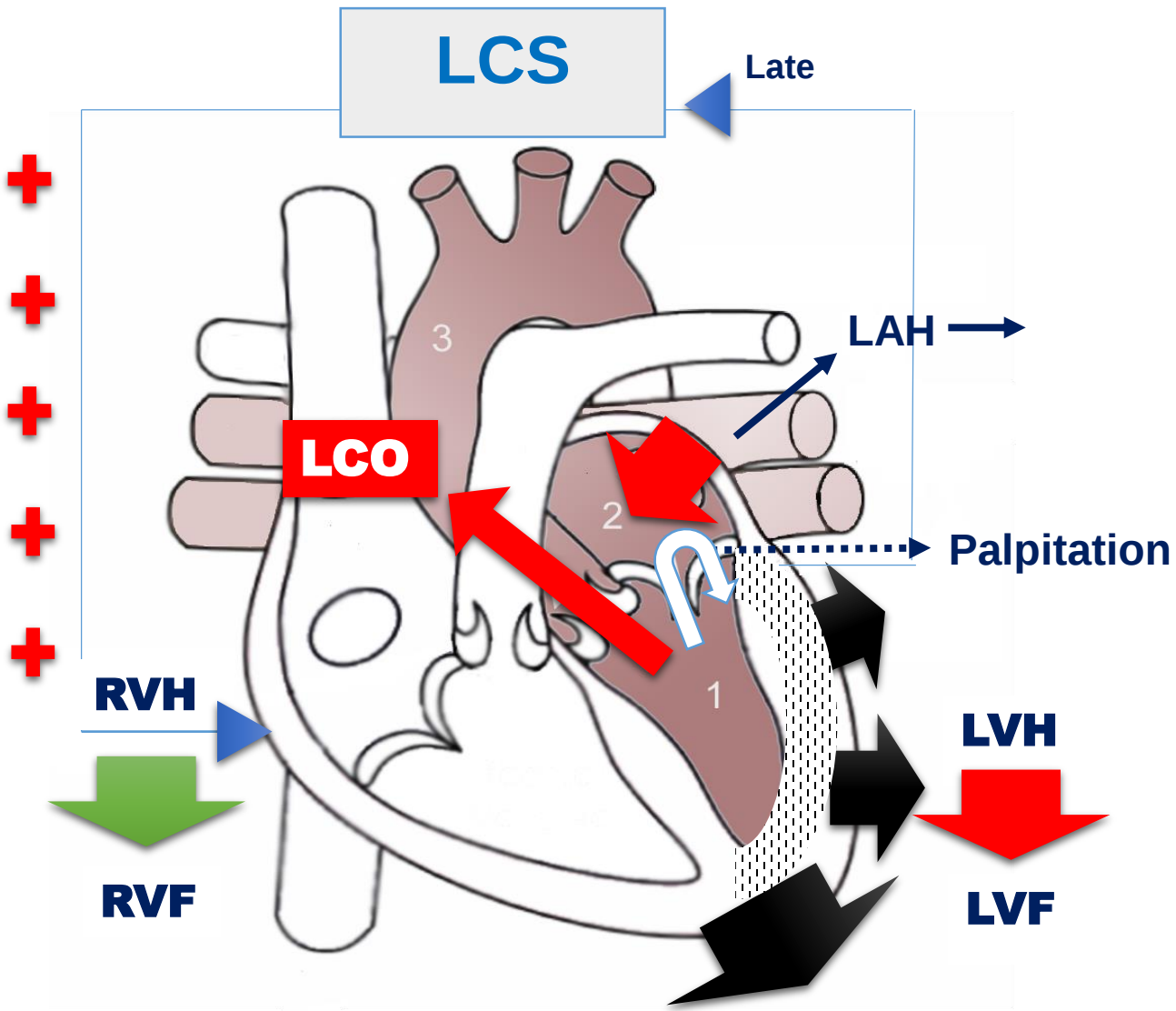


Aetiology

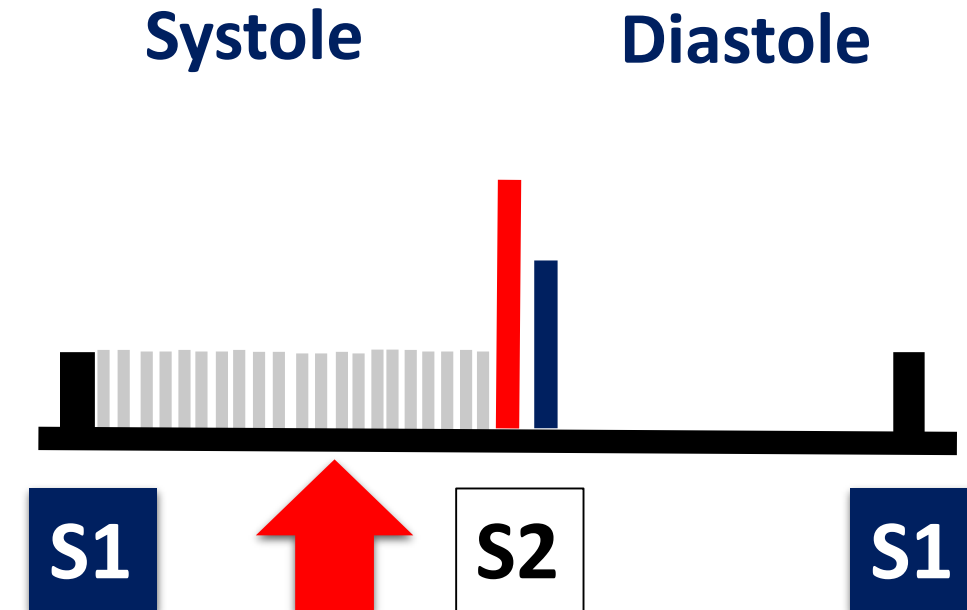
- ① Rheumatic heart disease
- ② Mitral Valve Prolapse (MVP)
- ③ SBE
- ④ Functional (in cases of LVF: due to Dilatation of MV ring)



Clinical Picture



Clinical Picture



Pan Systolic Murmur

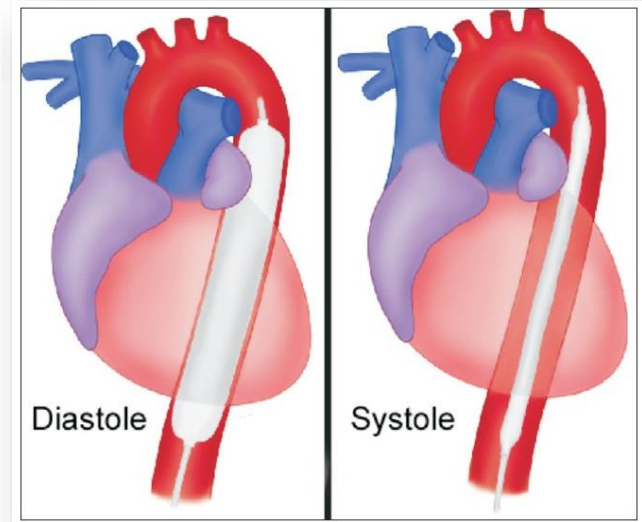
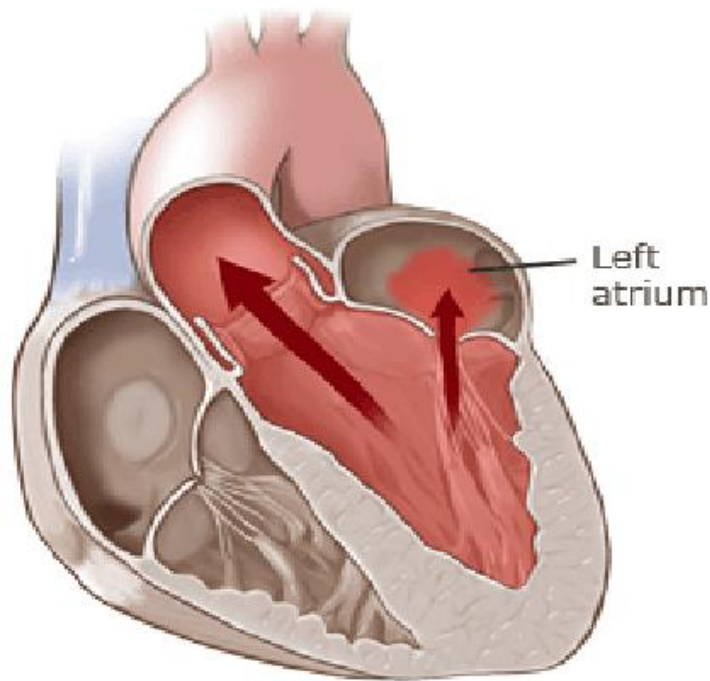
- 1- Apex
- 2- Radiates to axilla
- 3- High pitch
- 4- +++ by lying on left side

Special Investigations

- 1 ECG: LVH
- 2 X-Ray: LVH
- 3 Echo: **Diagnostic**

Treatment

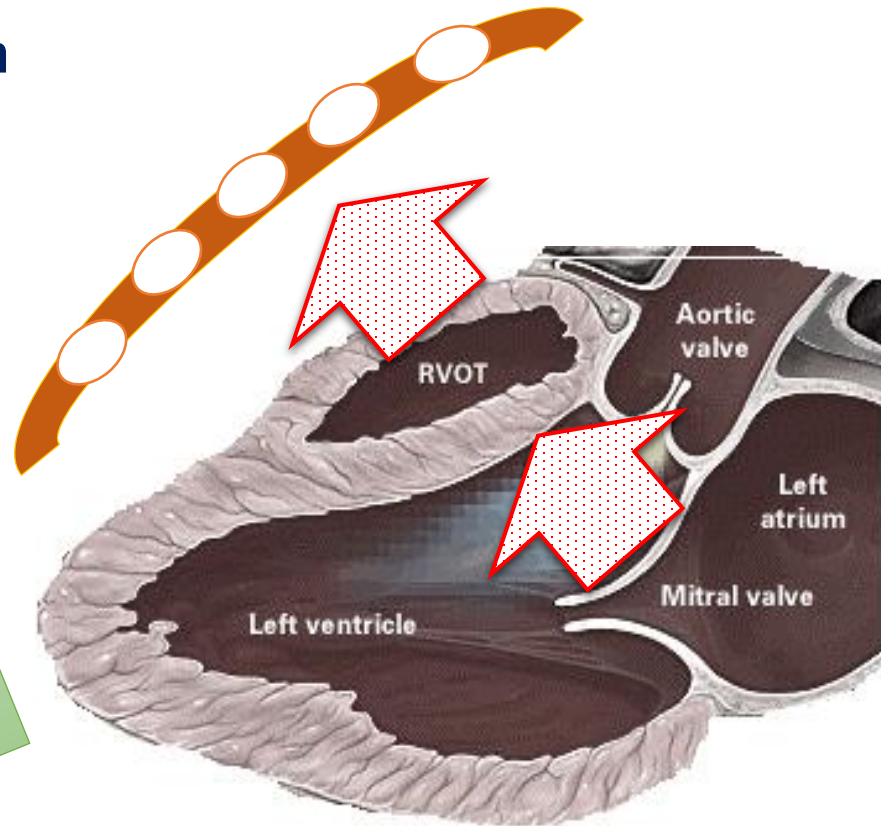
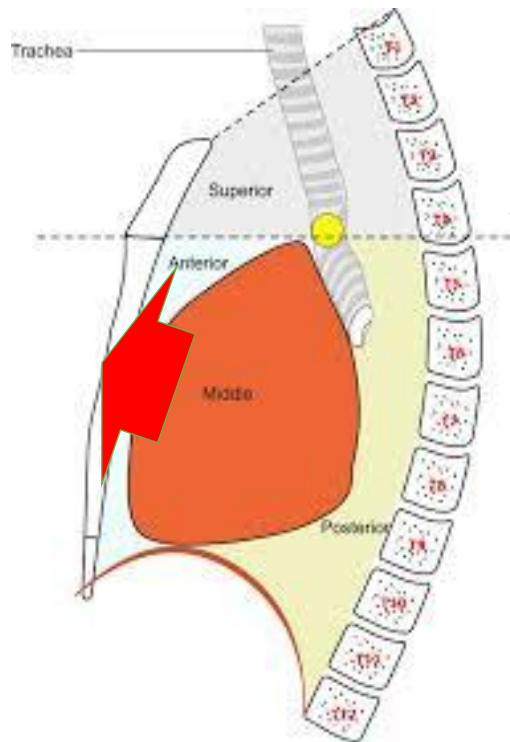
- ① Prophylaxis against Rheumatic Fever and SBE
- ② Surgery (Valve replacement)
- ③ In **Acute MR** vasodilator therapy or Intraaortic balloon counter pulsations may be useful to stabilize the patient by decreasing the afterload and thus allowing more blood to go to the Aorta.



- ④ In **Mitral Valve Prolapse** MVP decreasing contractility of the heart by beta blockers or Ca channel blockers may decrease the degree of Mitral Resurge.

Note

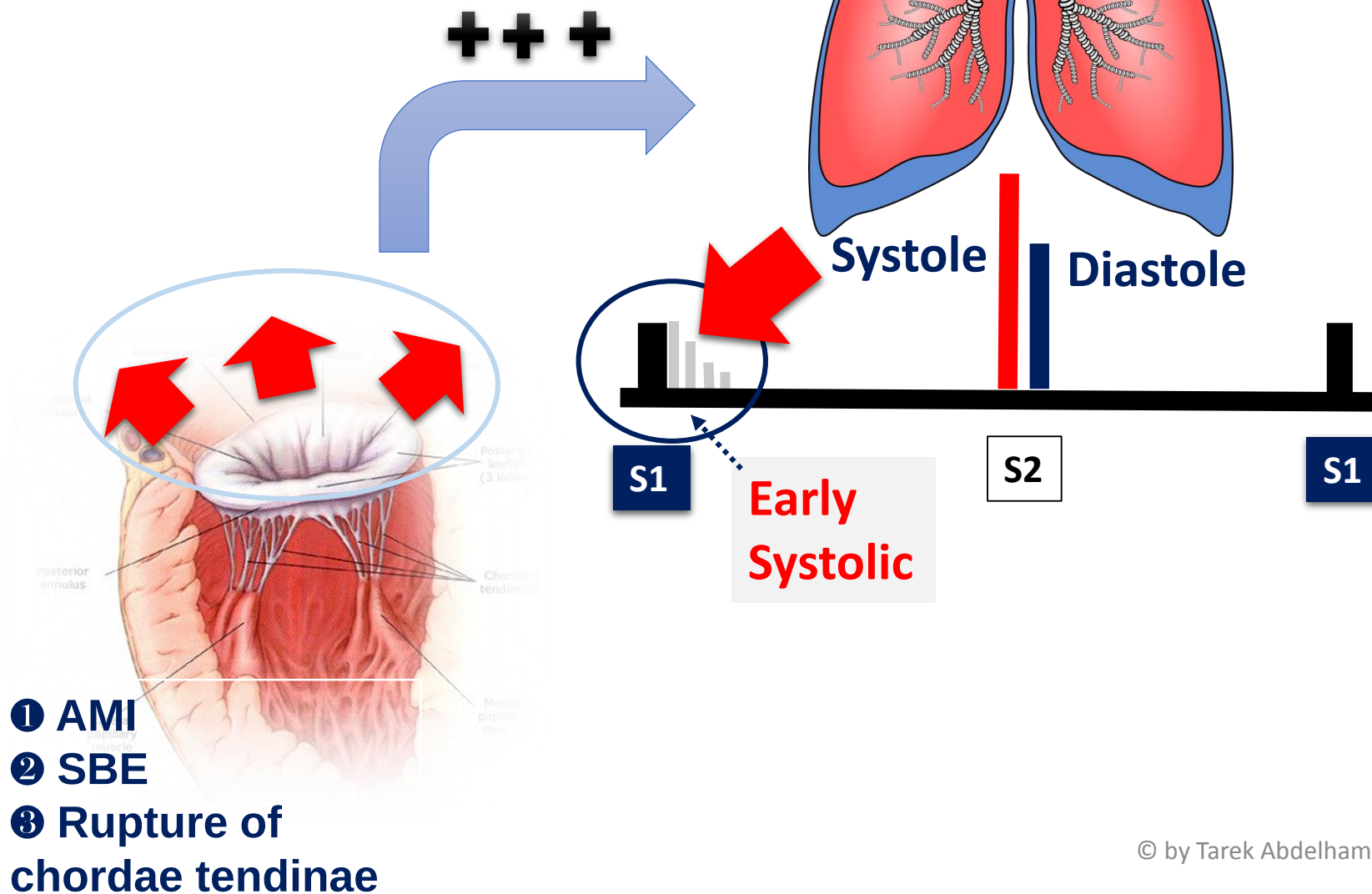
The expansions of the left atrium by the resurged blood may push the RV (which lies anterior to it) against the chest wall causing Parasternal pulsations of RV origin.



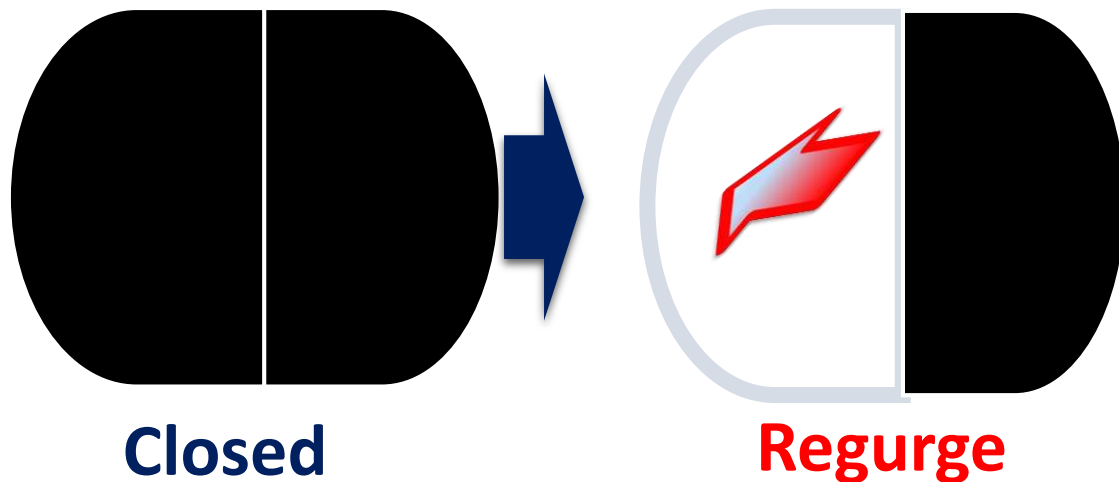
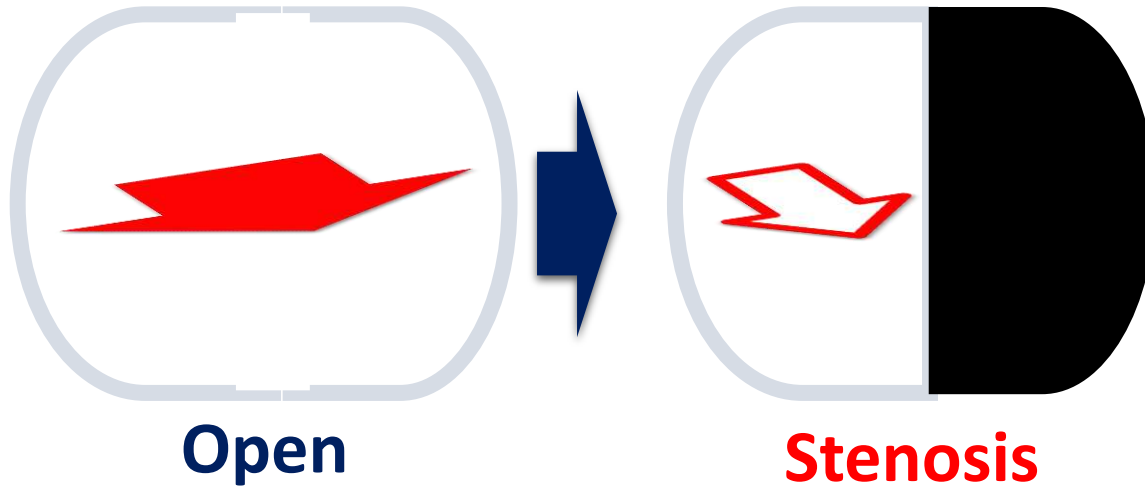
Note

Acute MR

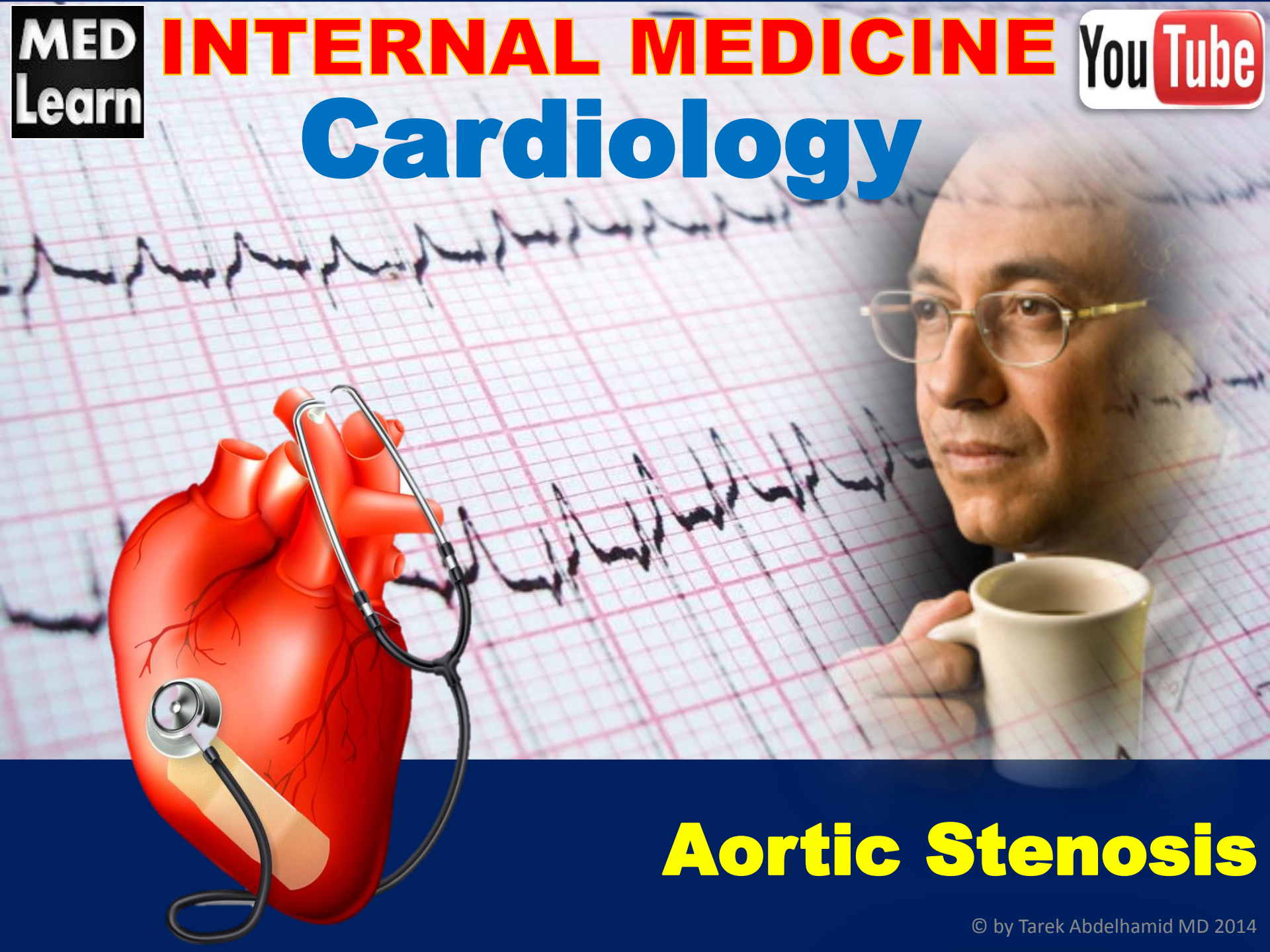
Acute Pulmonary Oedema



Double Mitral lesion?



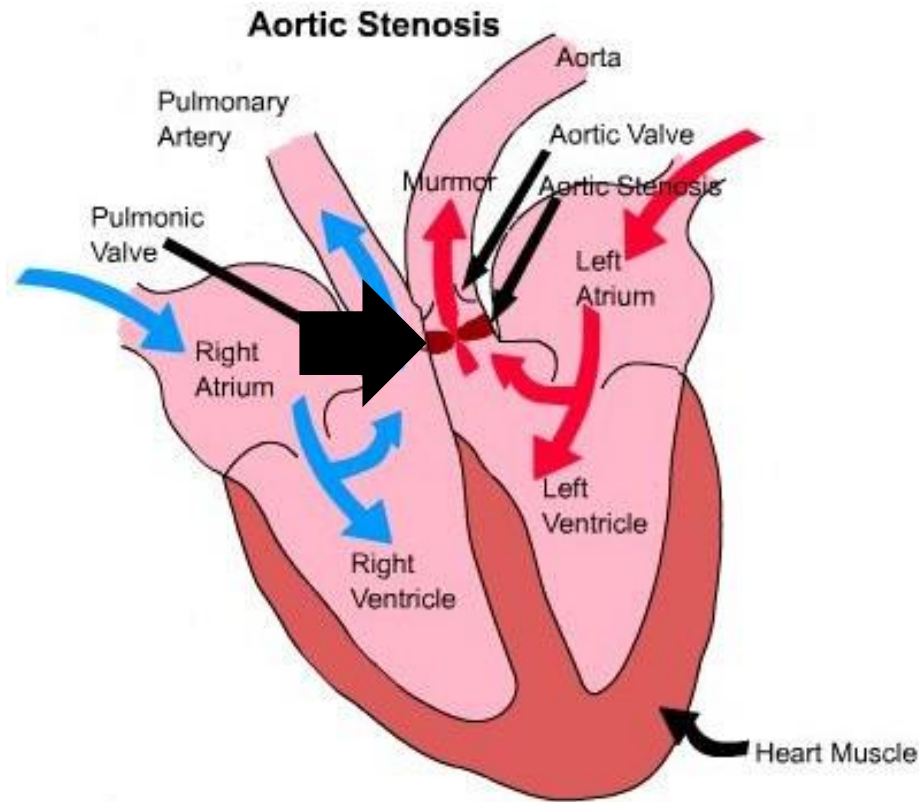
Cardiology



Aortic Stenosis

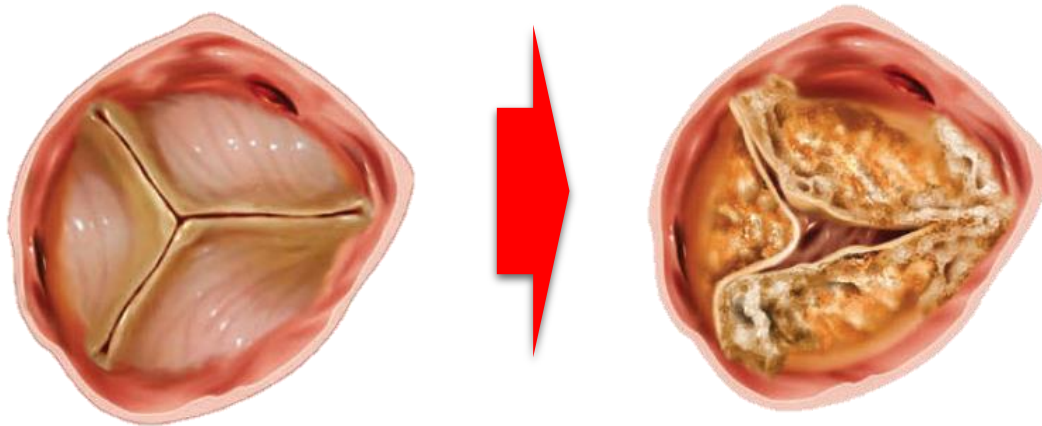
Definition

Inability of the **Aortic** Valve to open properly during Systole

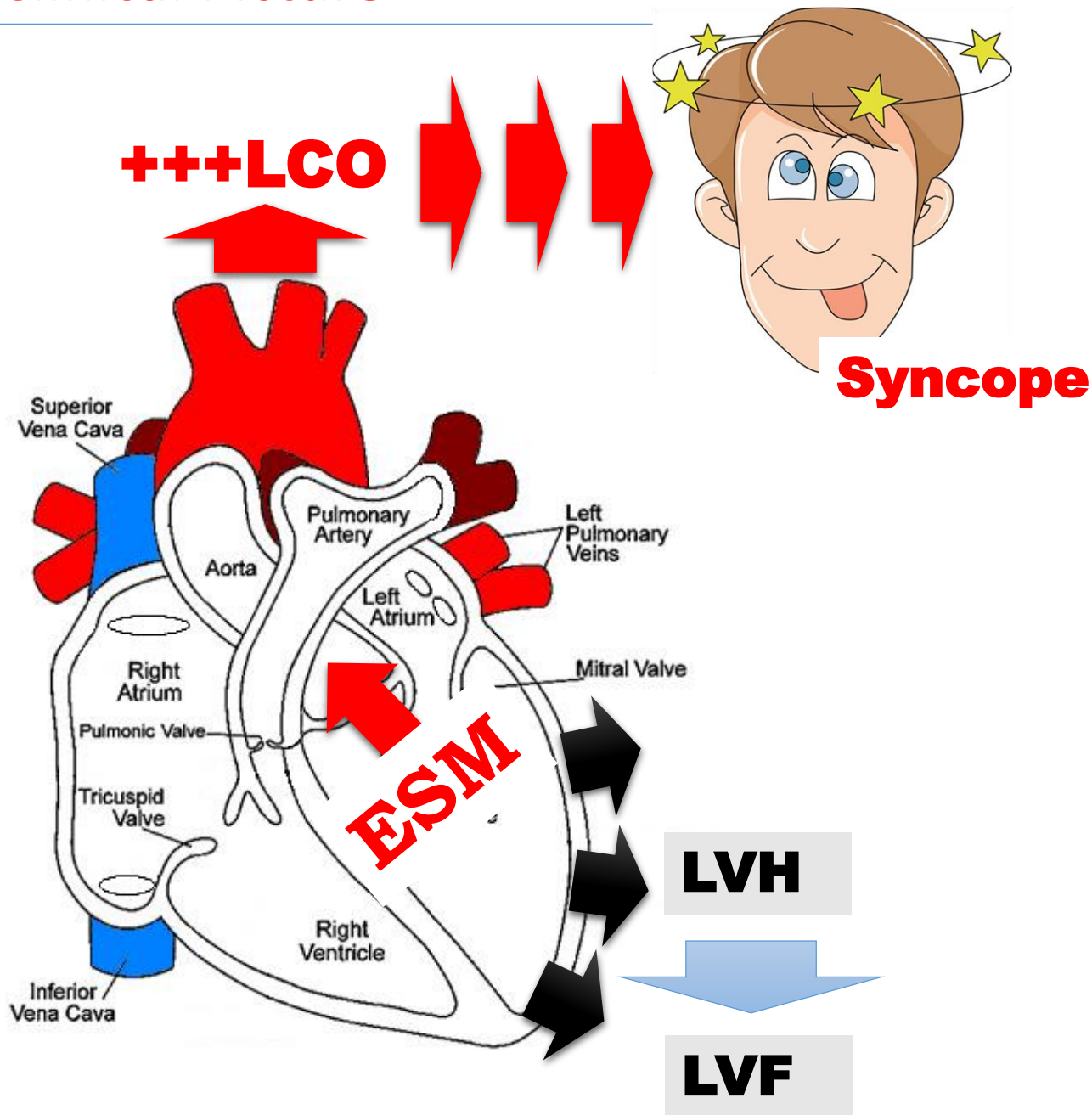


Aetiology

- ① **Senile AS** (Due to calcification)
- ② **Rheumatic**
- ③ **Big vegetation of SBE**
- ④ **Big lipid deposits in +++ Hyperlipidemia**

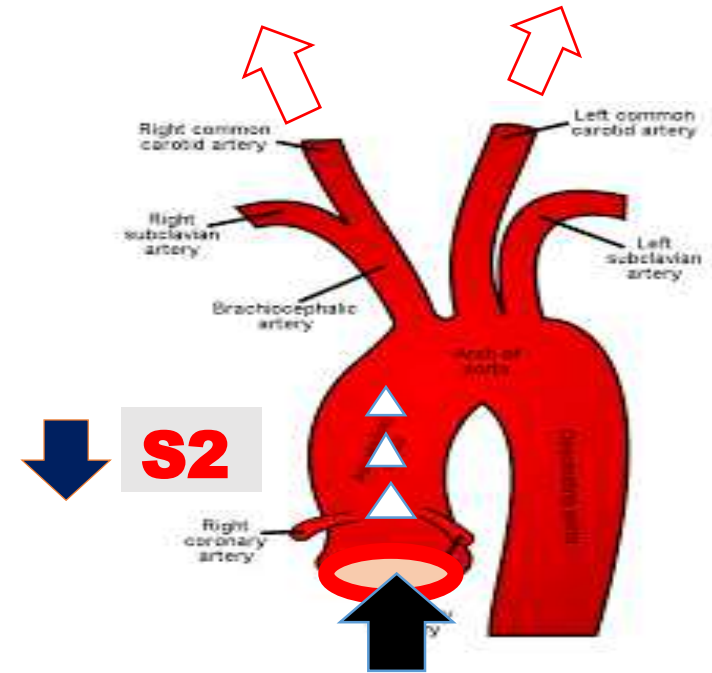
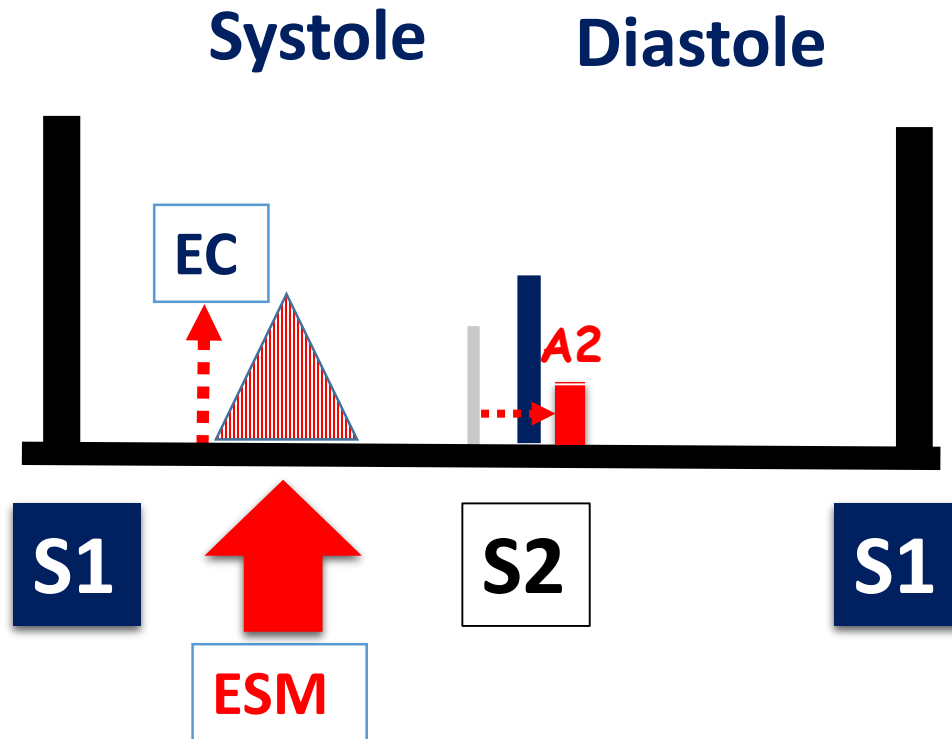


Clinical Picture



**Plateau
Pulse**

Auscultation

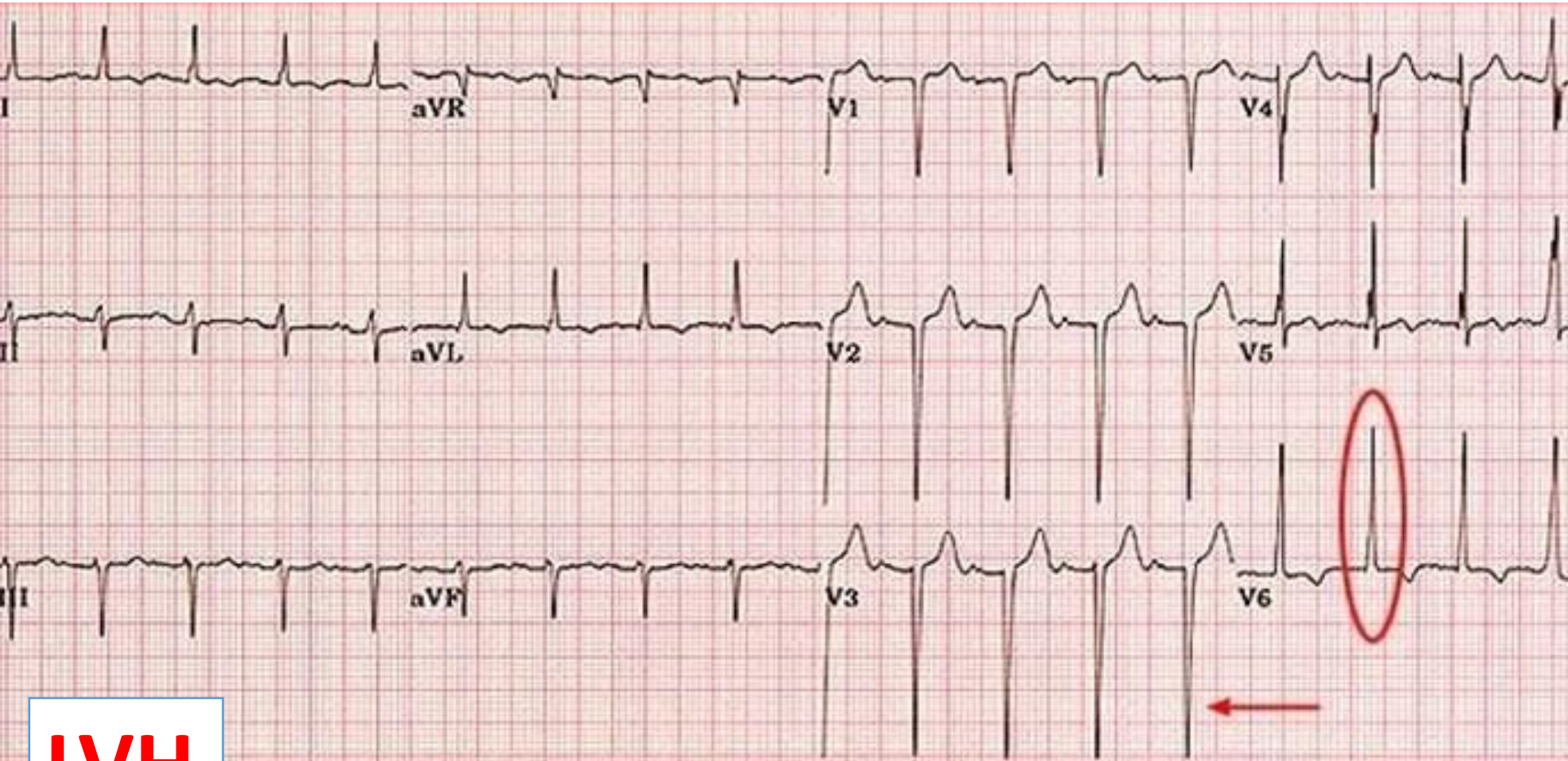


- ① Over the aortic area
- ② Radiates Up to carotids and down to lower left sternal border
- ③ Harsh murmur (due to +++ pressure) with a thrill
- ④ Increase by Leaning forward

Special Investigations

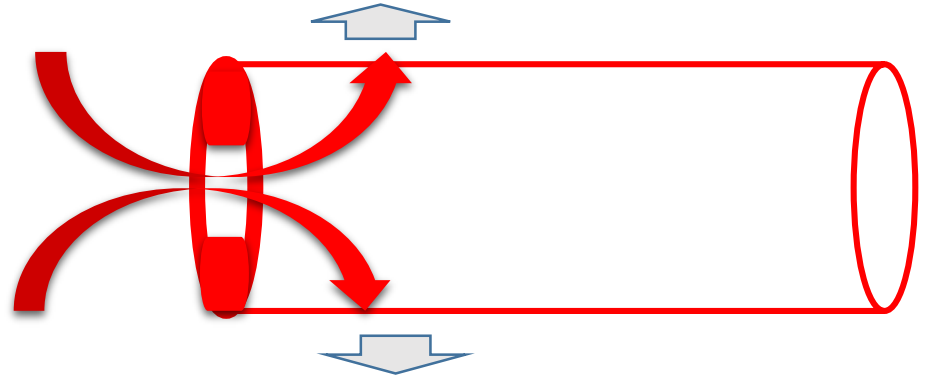
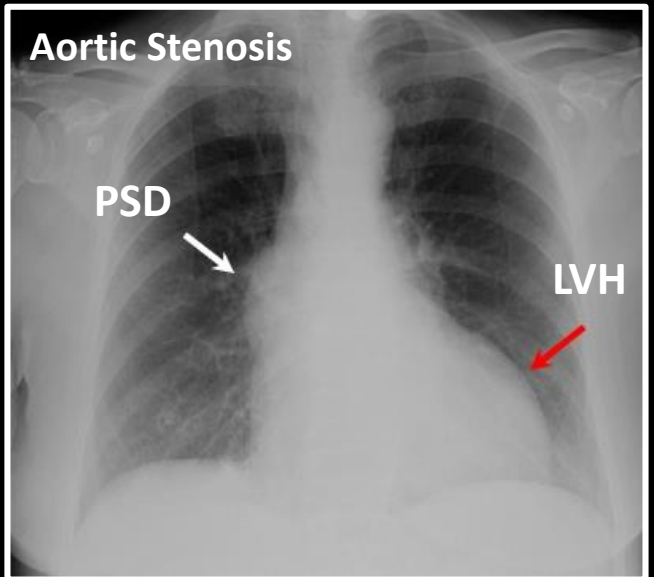
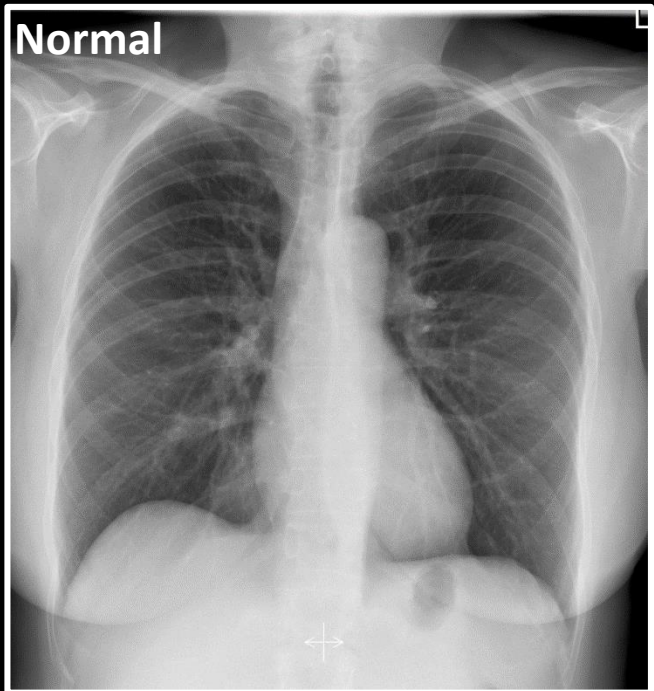
- 1 ECG: LVH
- 2 X-Ray: LVH
- 3 Echo: **Diagnostic**

Special Investigations



LVH

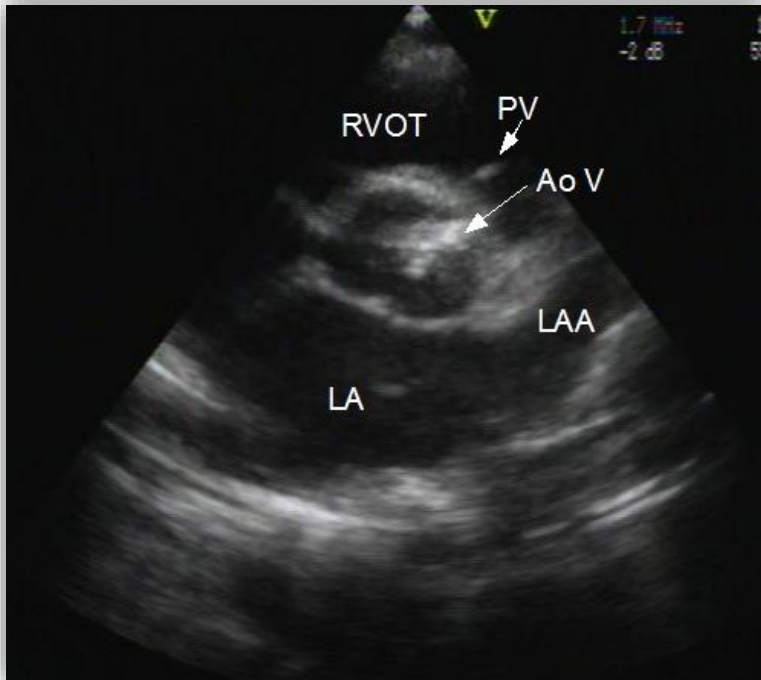
Special Investigations



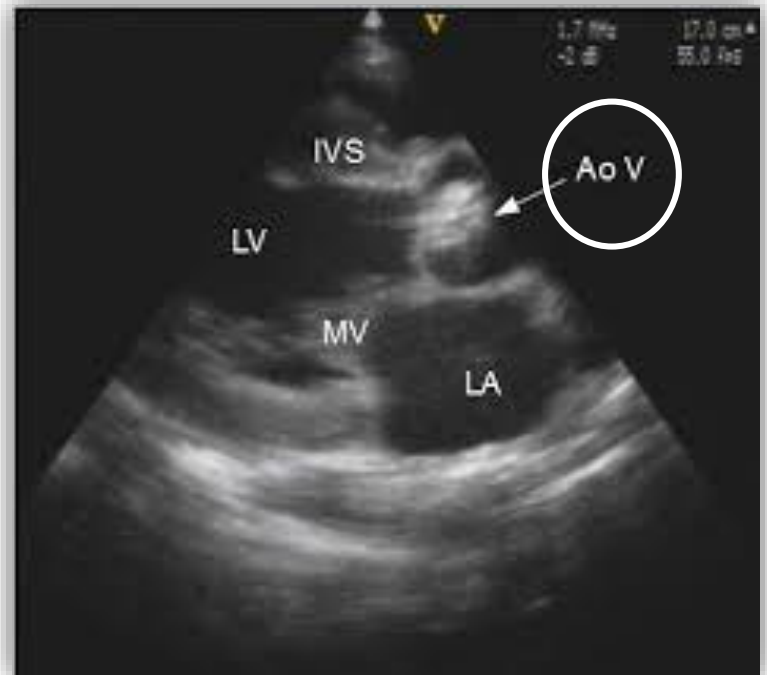
Post- Stenotic
dilatation

Special Investigations

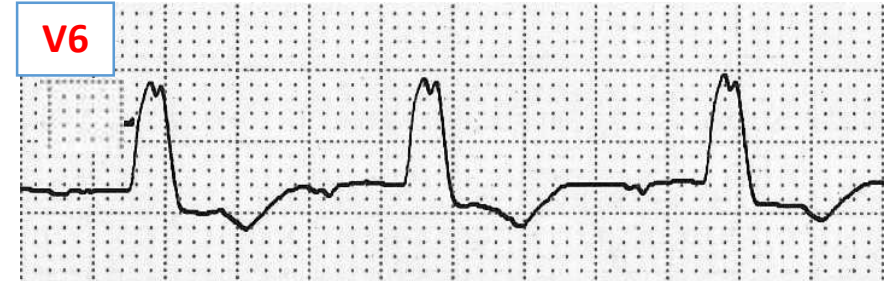
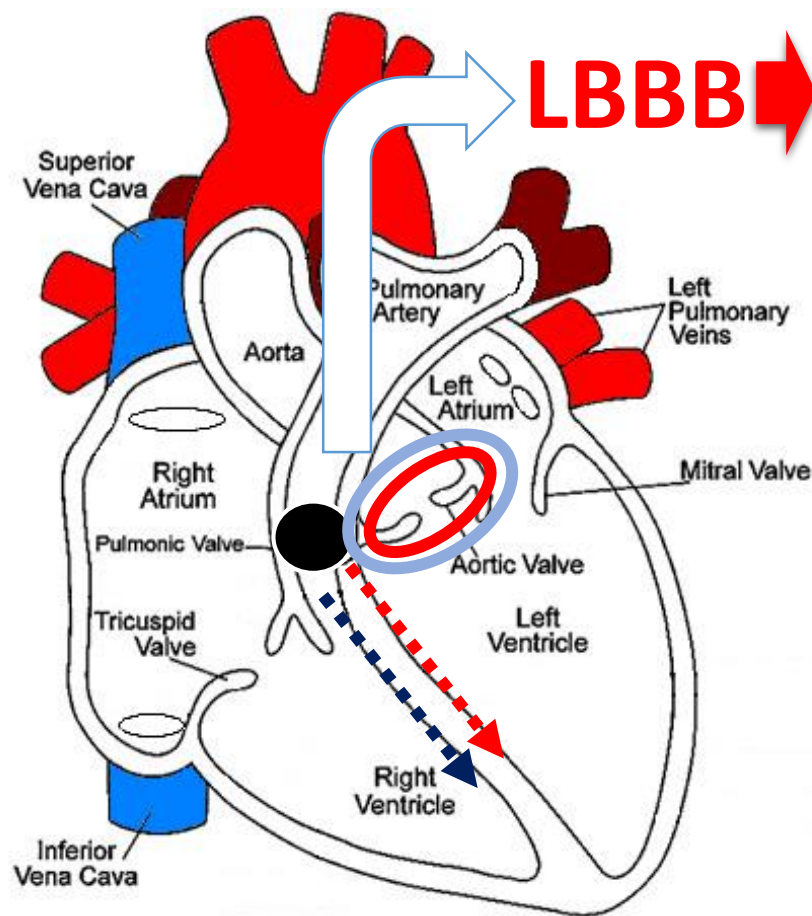
Normal Aortic Valve



Aortic Stenosis (AS)



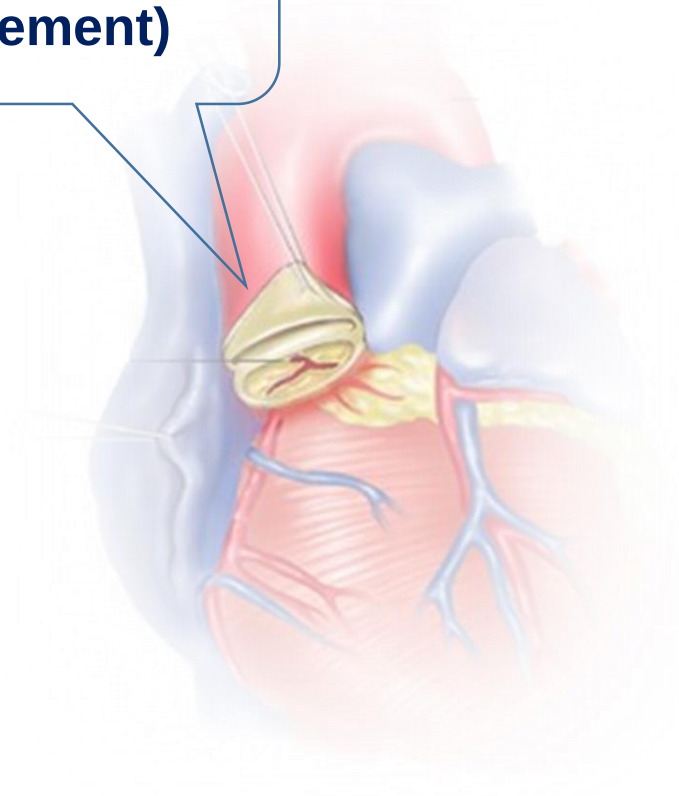
Note: LBBB in cases of AS



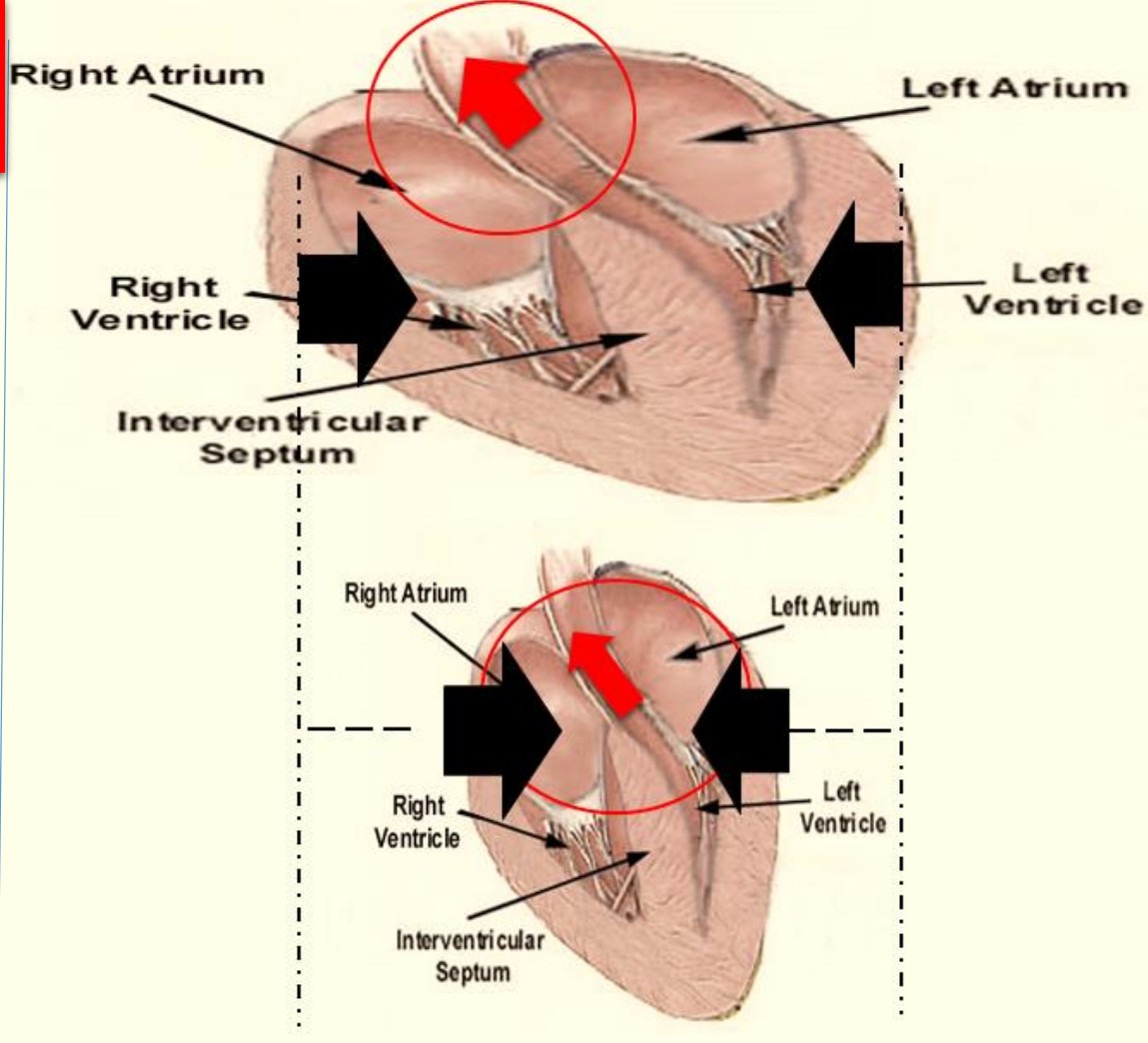
Extension of the calcification process around the Aortic Valve may affect the left bundle of the heart causing LBBB.

Treatment

**Main treatment is
Surgery (Valve
Replacement)**

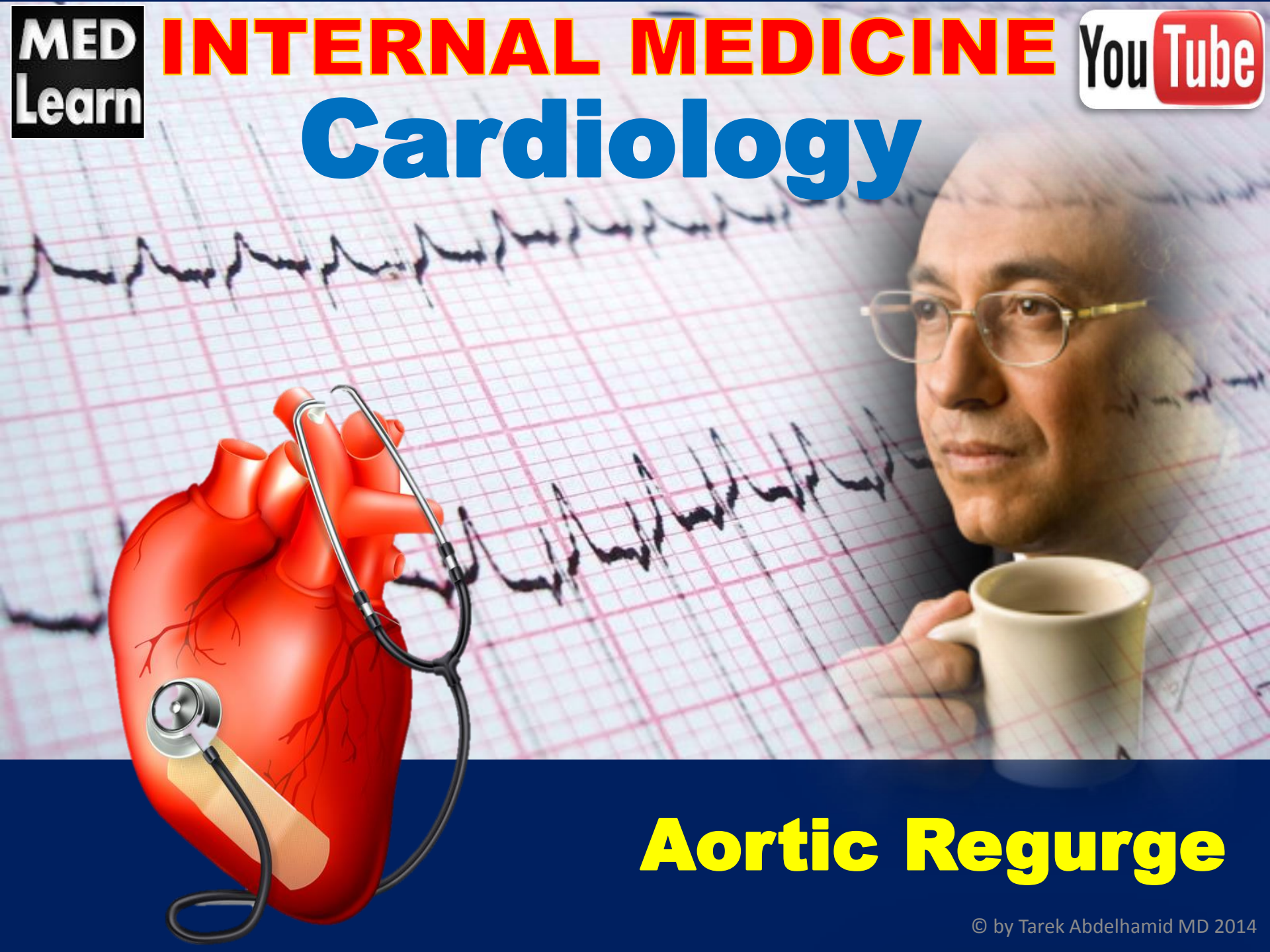


Subvalvular Aortic Stenosis



- ① Murmur decreases by elevating legs (+++ VR)
- ② The murmur increases by Valsalva maneuver (--- VR)
- ③ **NO** Ejection Click
- ④ ttt by Beta Blockers and Calcium channel antagonists

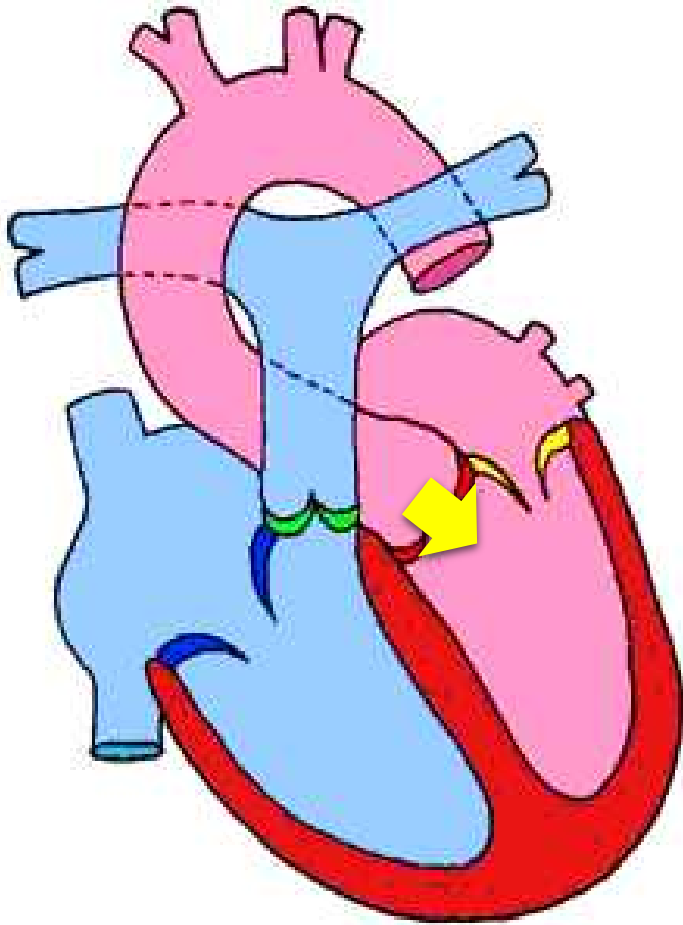
Cardiology



Aortic Regurge

Definition

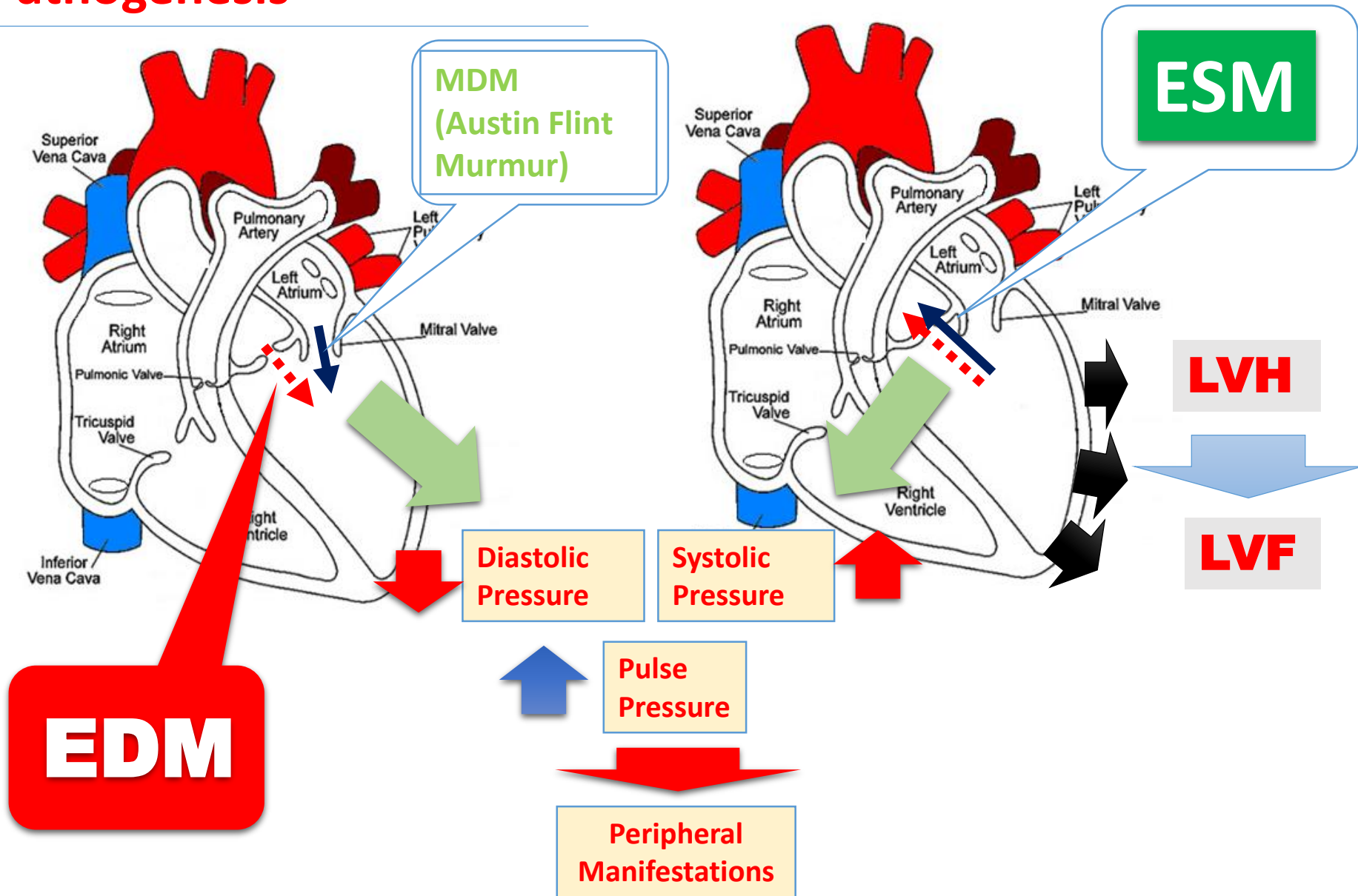
Inability of the **Aortic** Valve to close properly during diastole
(Incompetent Valve)



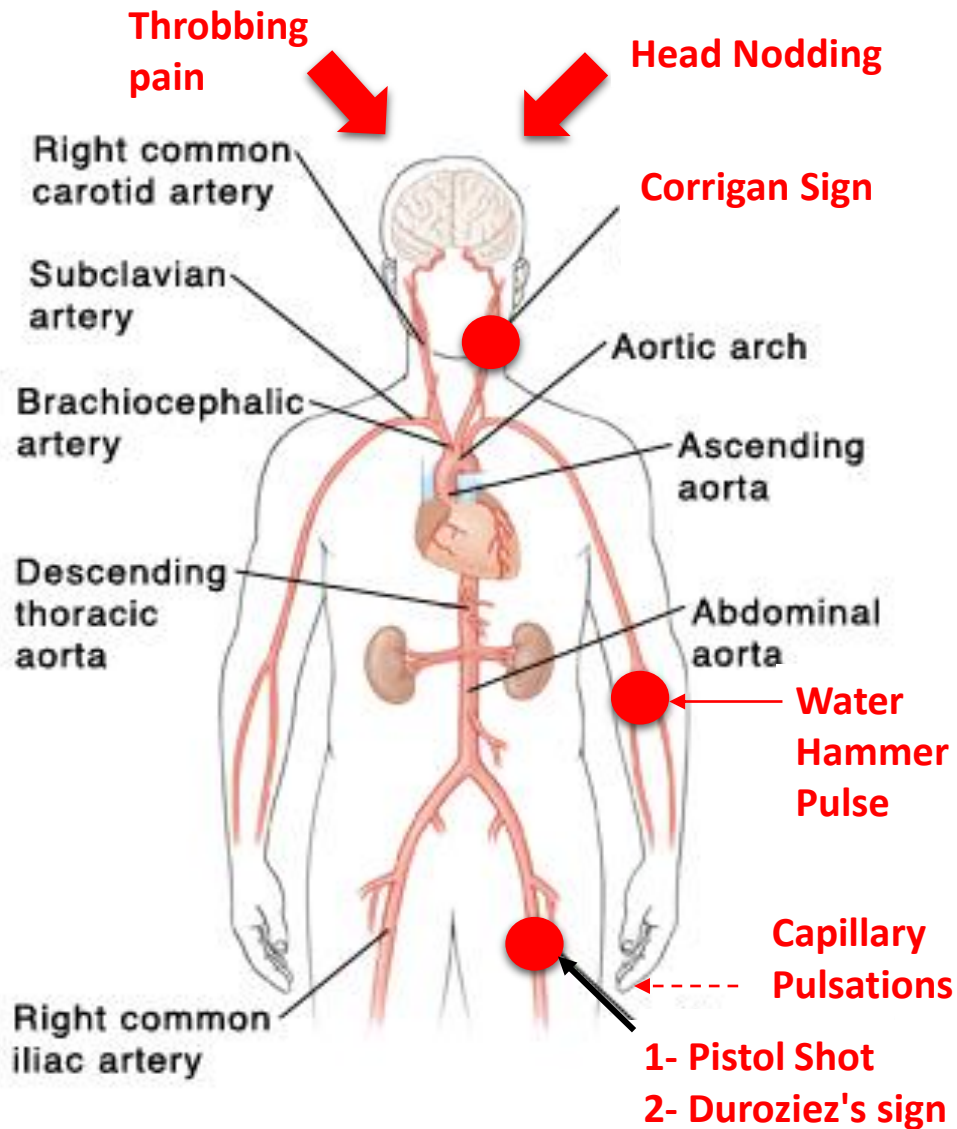
Aetiology

- ① Rheumatic
- ② SBE
- ③ Marfan Syndrome
- ④ SLE
- ⑤ Ankylosing Spondylitis
- ⑥ Ulcerative Colitis
- ⑦ Associated with VSD
- ⑧ Functional

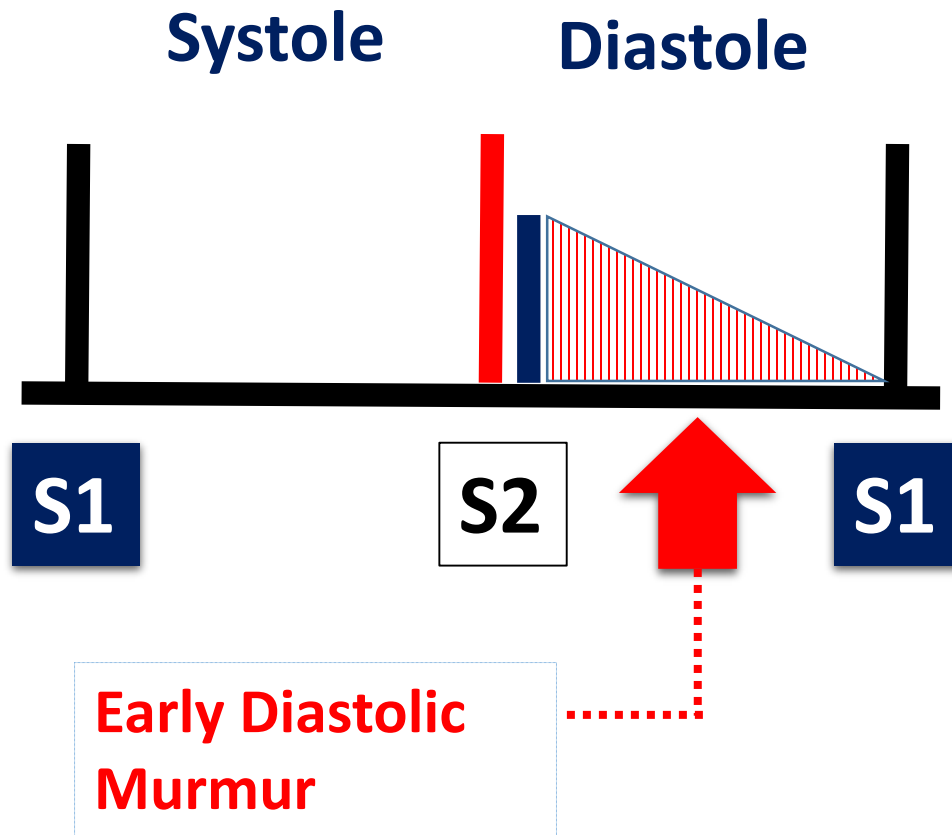
Pathogenesis



Peripheral Manifestations



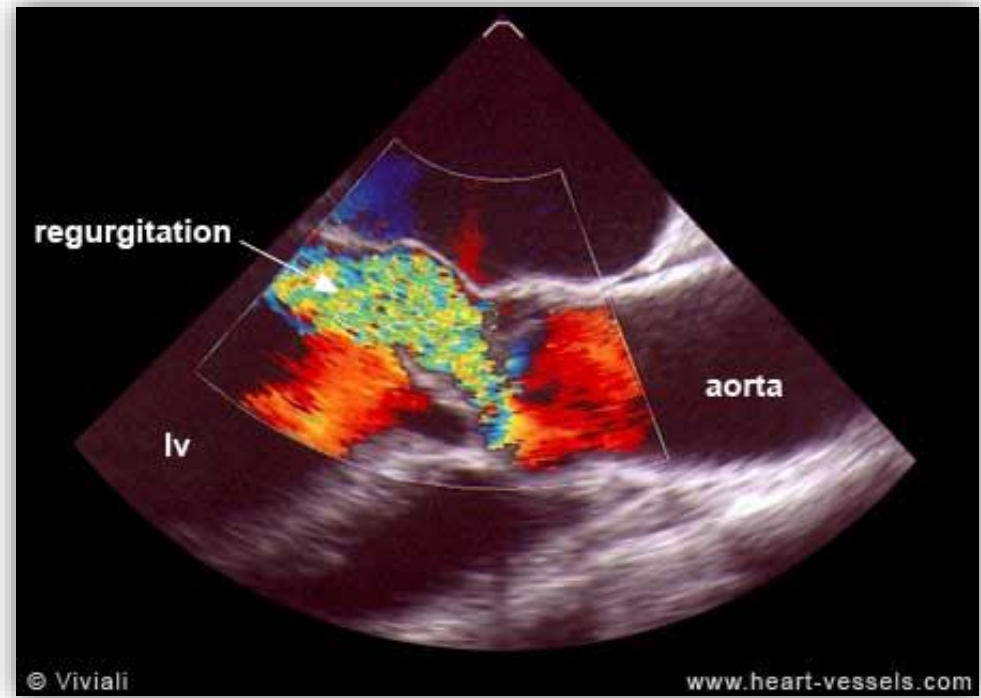
Auscultation



- ① Over the aortic area
- ② Radiates to lower left sternal border
- ③ Decrescendo murmur
- ④ Blowing in character
- ⑤ High pitch
- ⑥ Increase by leaning forward

Special Investigations

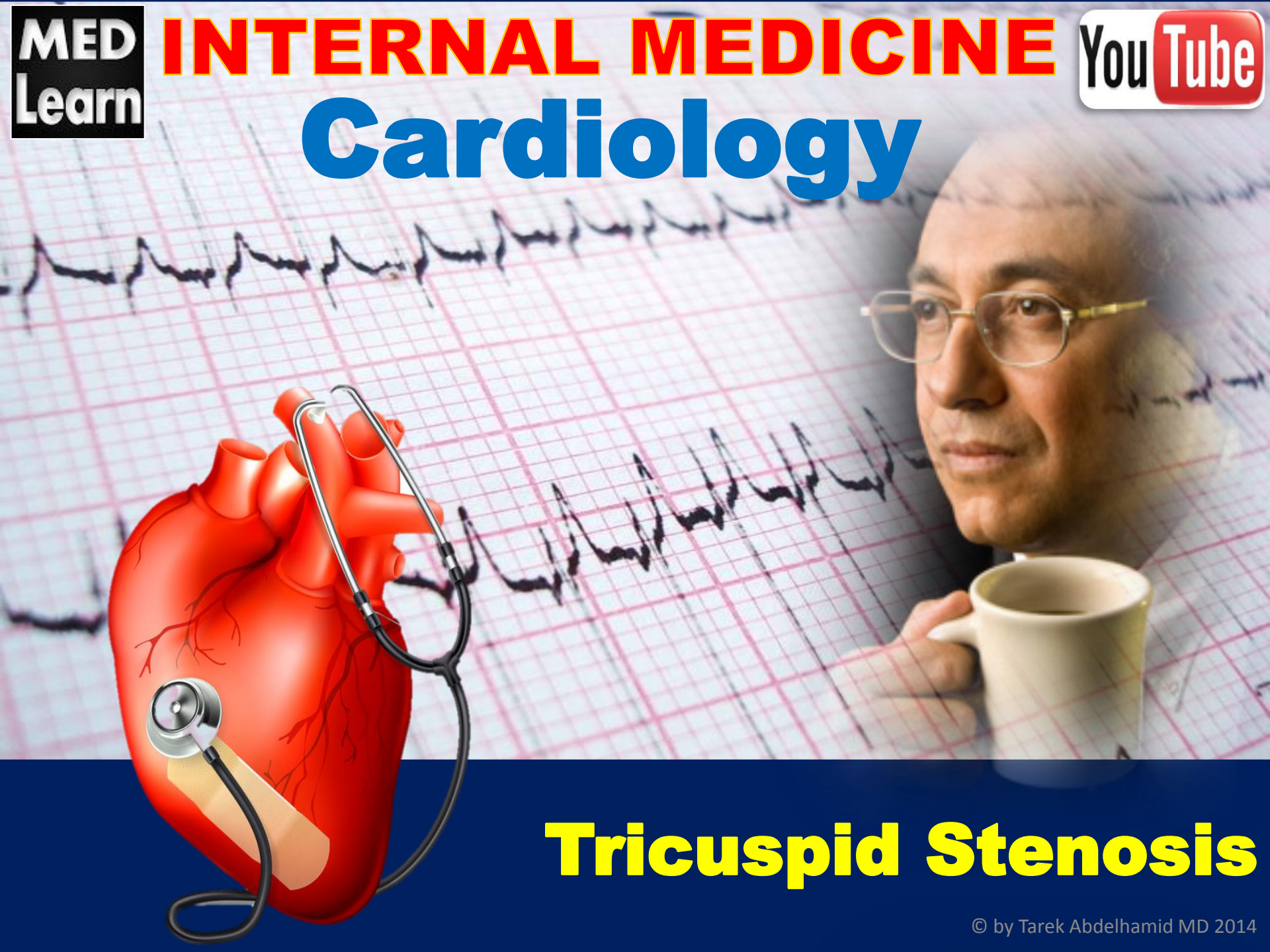
- ① ECG: LVH
- ② X-Ray: LVH
- ③ Echo: **Diagnostic**



Treatment

- ① Main treatment is Surgery (Valve Replacement)
- ② Prophylaxis for Rheumatic Fever
- ③ Prophylaxis for SBE

Cardiology



Tricuspid Stenosis

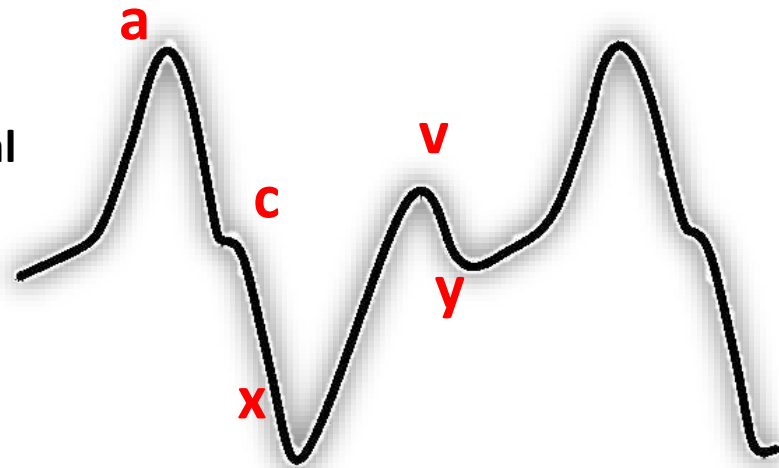
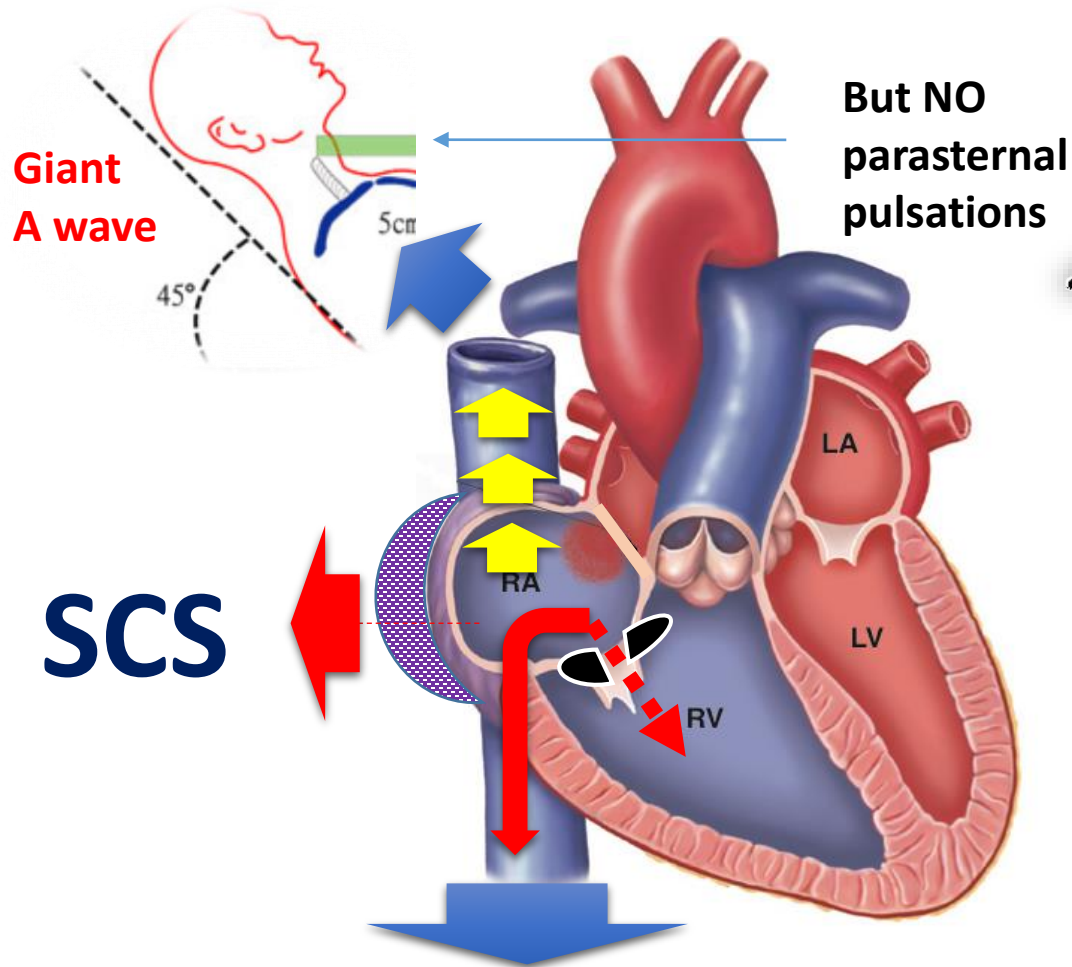
Definition

Inability of the **Tricuspid** Valve to open properly during Systole.

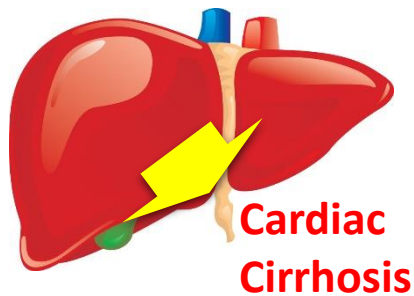
Aetiology

- ① Rheumatic
- ② Big Vegetations of SBE
- ③ Carcinoid Syndrome

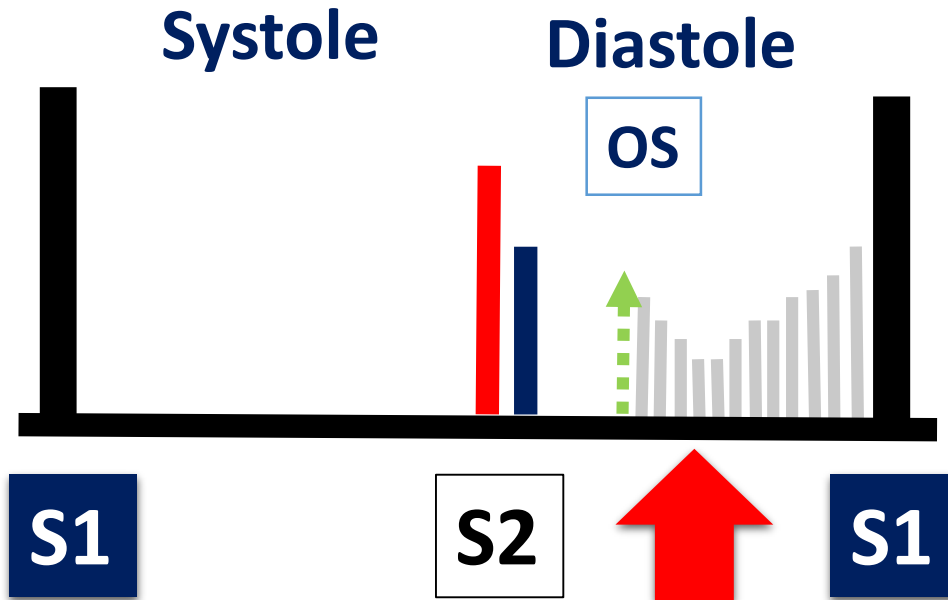
Clinical Picture



Presystolic Pulsations



Auscultation



MDM with PSA

Lower Left Sternal
Border

By Inspiration

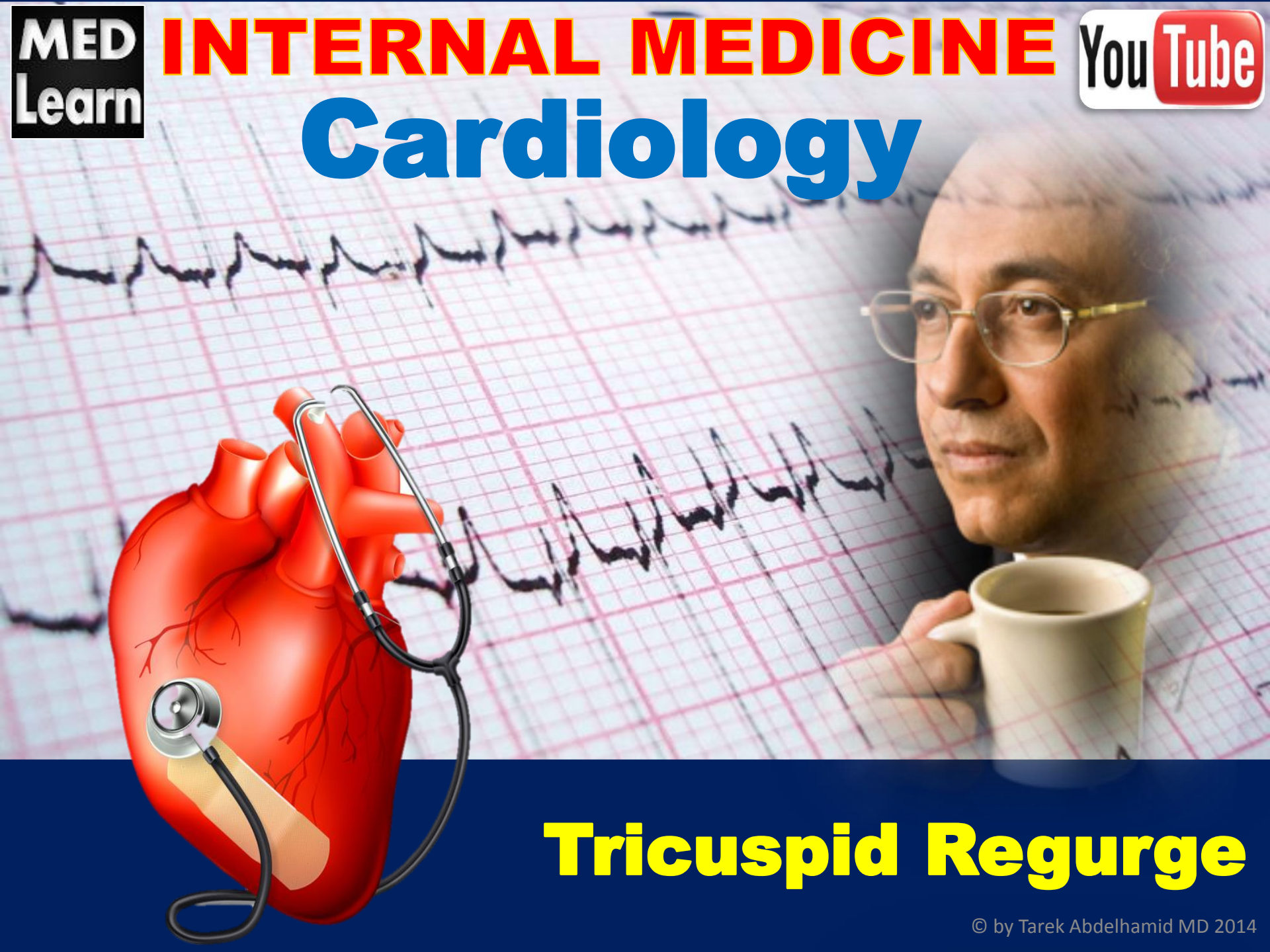
Special Investigations

- ① ECG: LVH
- ② X-Ray: LVH
- ③ Echo: **Diagnostic**

Treatment

Valve Replacement

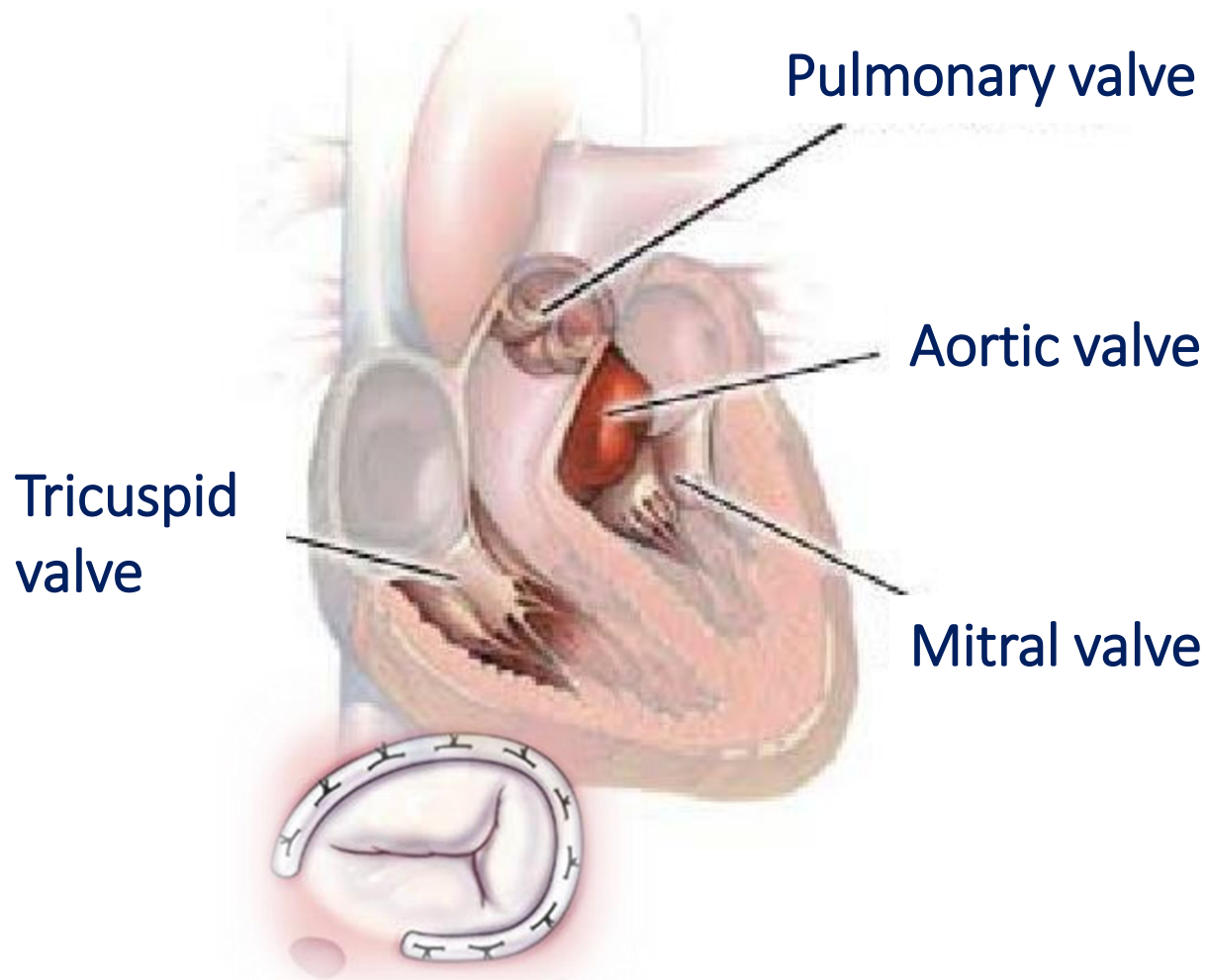
Cardiology



Tricuspid Regurge

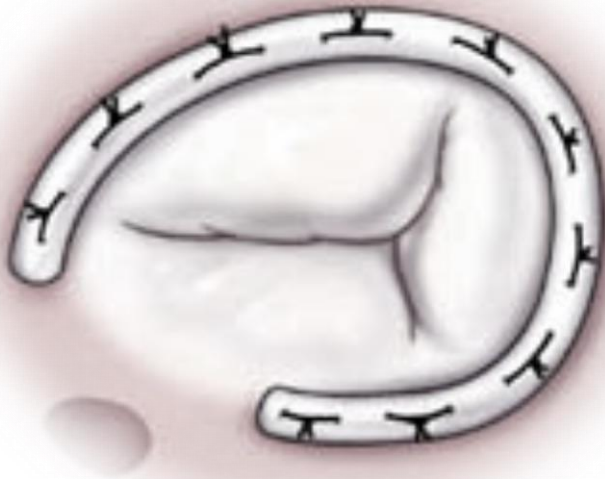
Definition

Inability of the TV to close properly during systole (Tricuspid Incompetence)



Aetiology

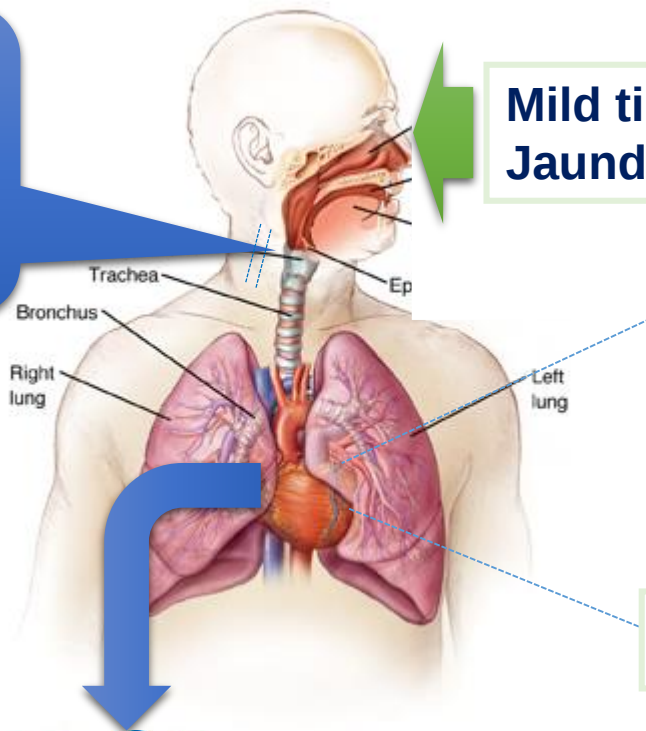
- ① RSHF (**Functional**)
- ② Papillary Muscle Dysfunction (**Inferior Wall AMI**)
- ③ SLE
- ④ Carcinoid Syndrome
- ⑤ SBE (epically in **Drug Addicts**)



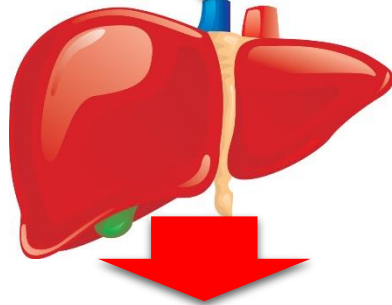
Clinical Picture

**Systolic
Expansion
Of Neck
Veins**

**Mild tinge of
Jaundice**

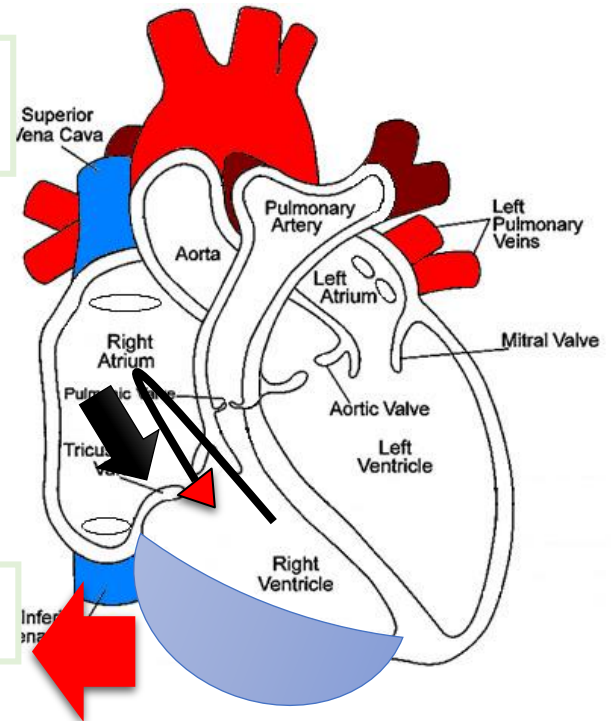


**Systolic
Expansion**

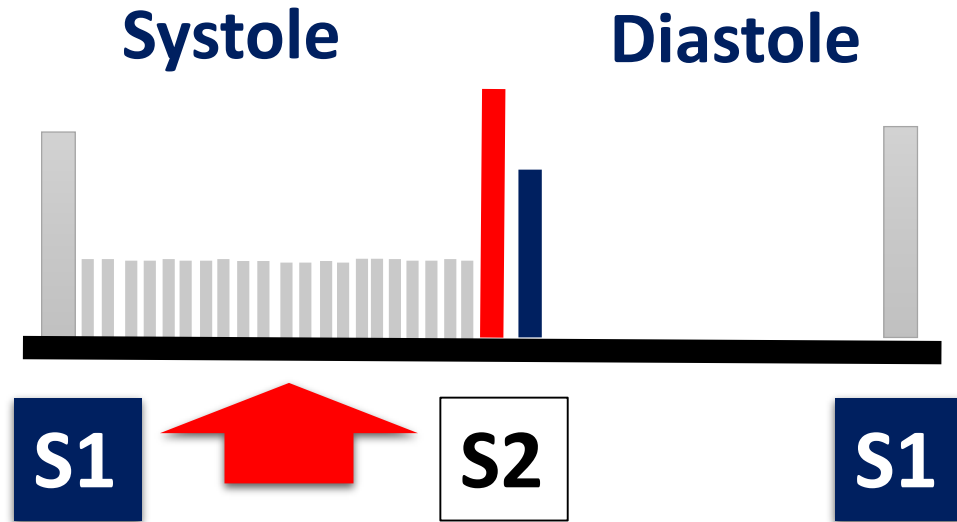


RVH

RVF



Auscultation

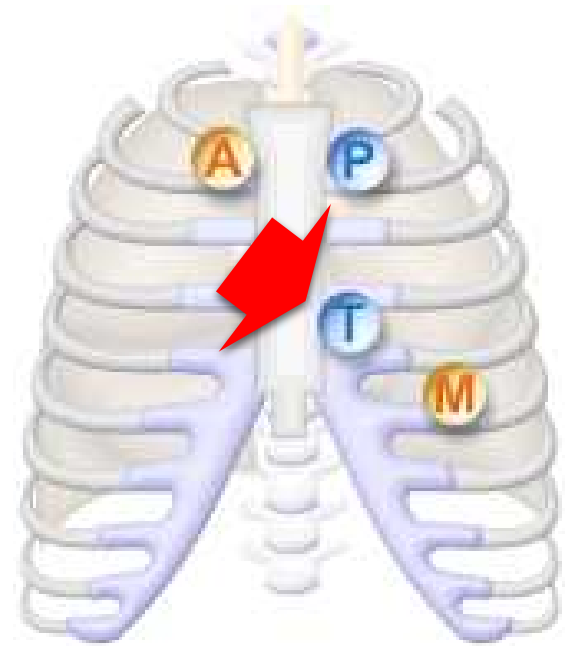


Pan Systolic Murmur

**Over the
tricuspid
area**



Apex



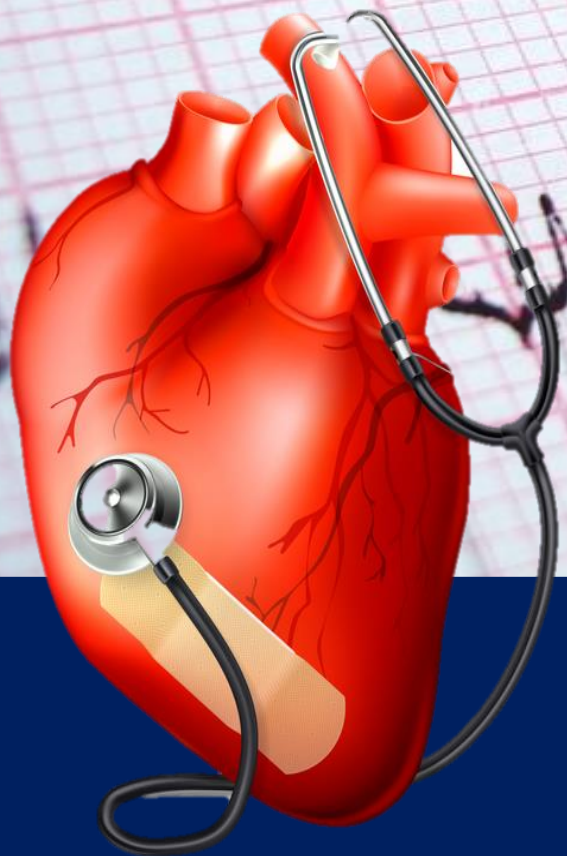
Special Investigations

- ① ECG: RVH
- ② X-Ray: RVH
- ③ Echo: **Diagnostic**

Treatment

Treatment is mainly surgical

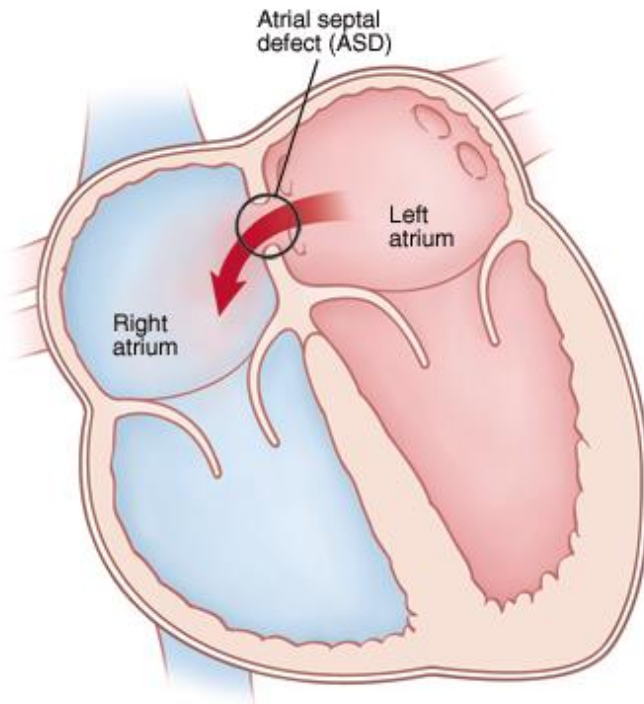
Cardiology



ASD

Definition

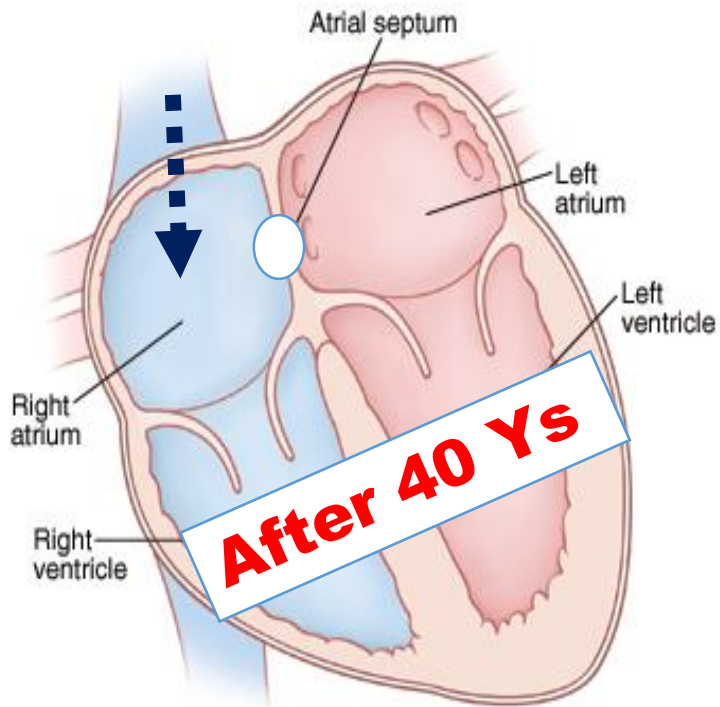
ASD is a defect in the interatrial septum



Aetiology

Most likely Congenital

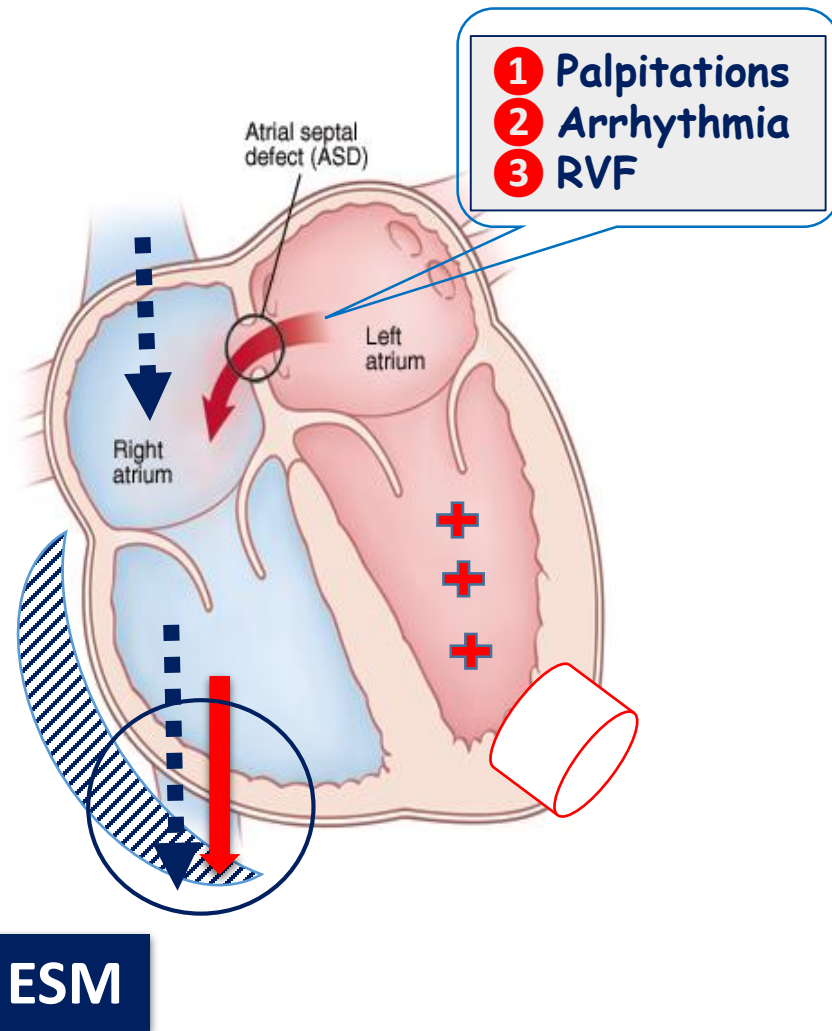
Clinical Picture



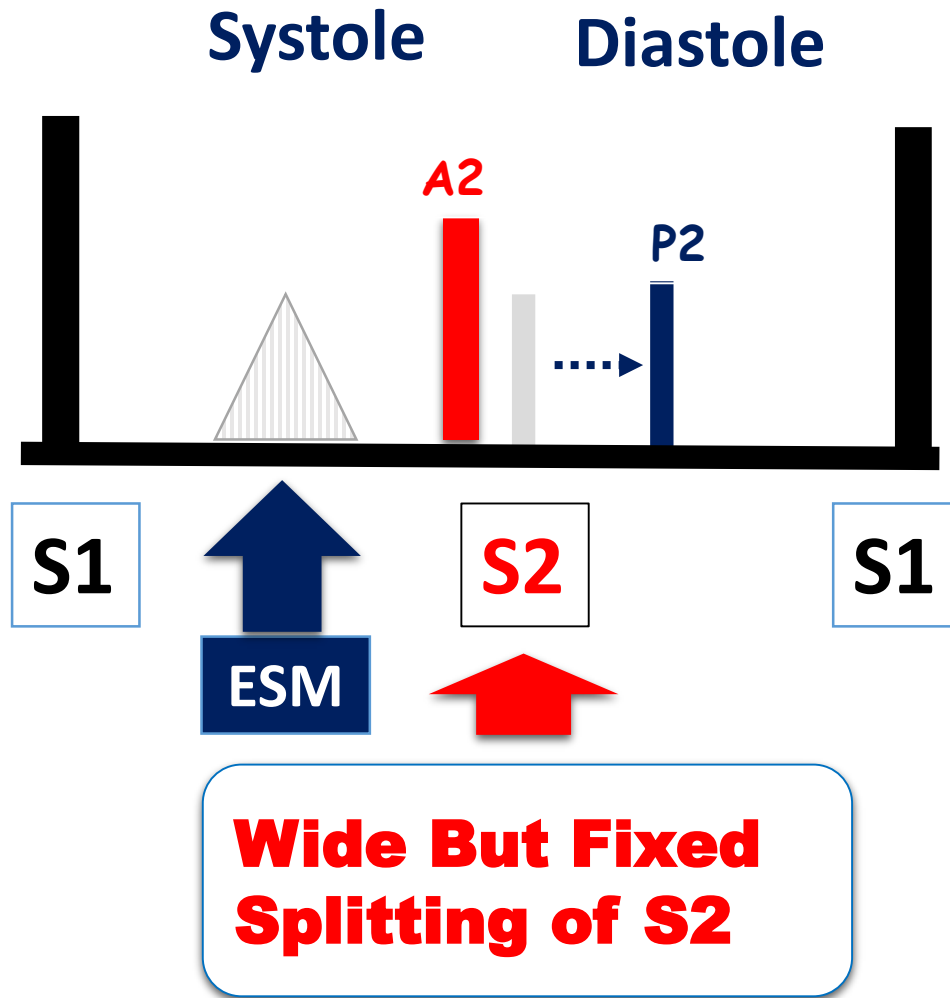
After 40 Ys

**Asymptomatic
For Years**

After 40 Ys due to:
1- Increase BP
2- Diminished LV Compliance



Auscultation

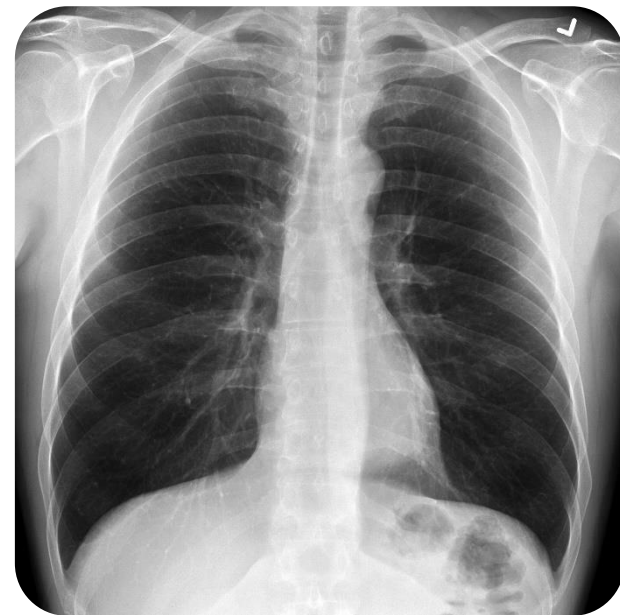


Special Investigations

① Right Ventricular conduction abnormalities may be seen on ECG



②  Lung vascularity on Chest X-Ray



Normal



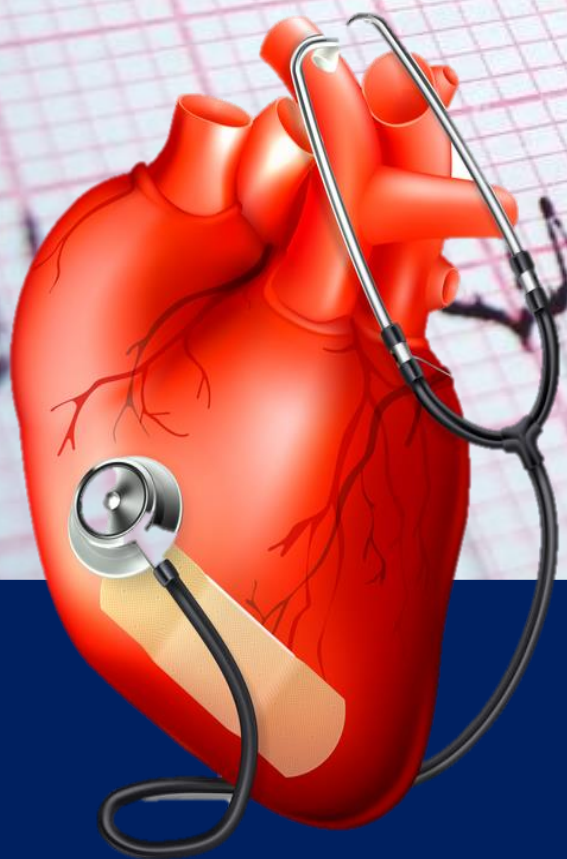
ASD

③ Echo-Doppler confirms the Diagnosis

Treatment

Surgery (if Symptomatic)

Cardiology



VSD

تحذير هام

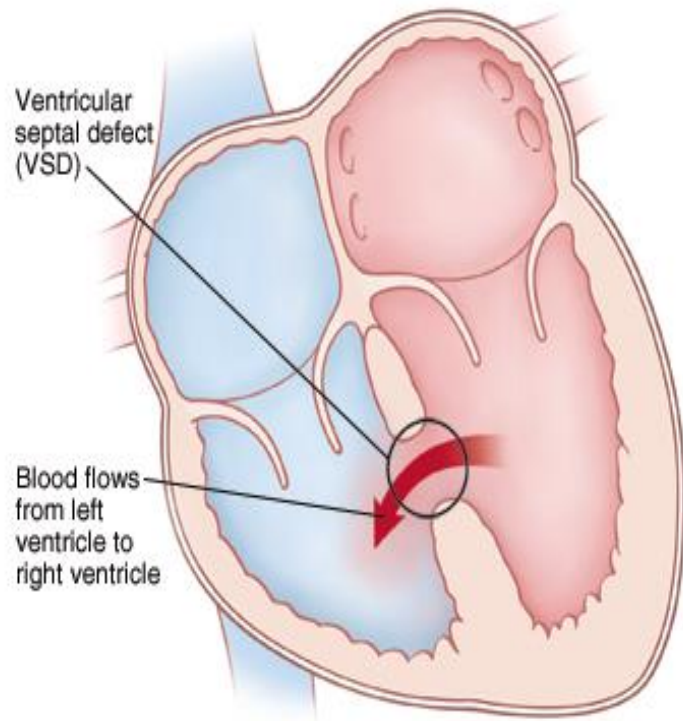
هذا الفيديو و جميع الرسوم التوضيحية المتاحة فى هذا الموقع هم تحت حماية قانون الملكية الفكرية

هذا الفيديو و جميع الرسوم التوضيحية المتاحة فى هذا الموقع هم تحت حماية قانون الملكية الفكرية و لا يحق لأحد إستخدامه أو إستنساخه أو إعادة طبعه أو طبع أجزاء منه بدون إذن مسبق من الدكتور طارق عبد الحميد (أو من ينوب عنه قانونياً). و من يتعدى على الملكية الفكرية المذكورة فسوف يتعرض للمسائلة القضائية عن جريمة إنتهاك حقوق الملكية الفكرية. و إستخدام الفيديوهات و المادة العلمية المذكورة مكفول فقط لمشتري واحد فقط و لأخذ تصريح إستخدام لأكثر من شخص برجاء الإتصال بالدكتور طارق عبد الحميد



Definition

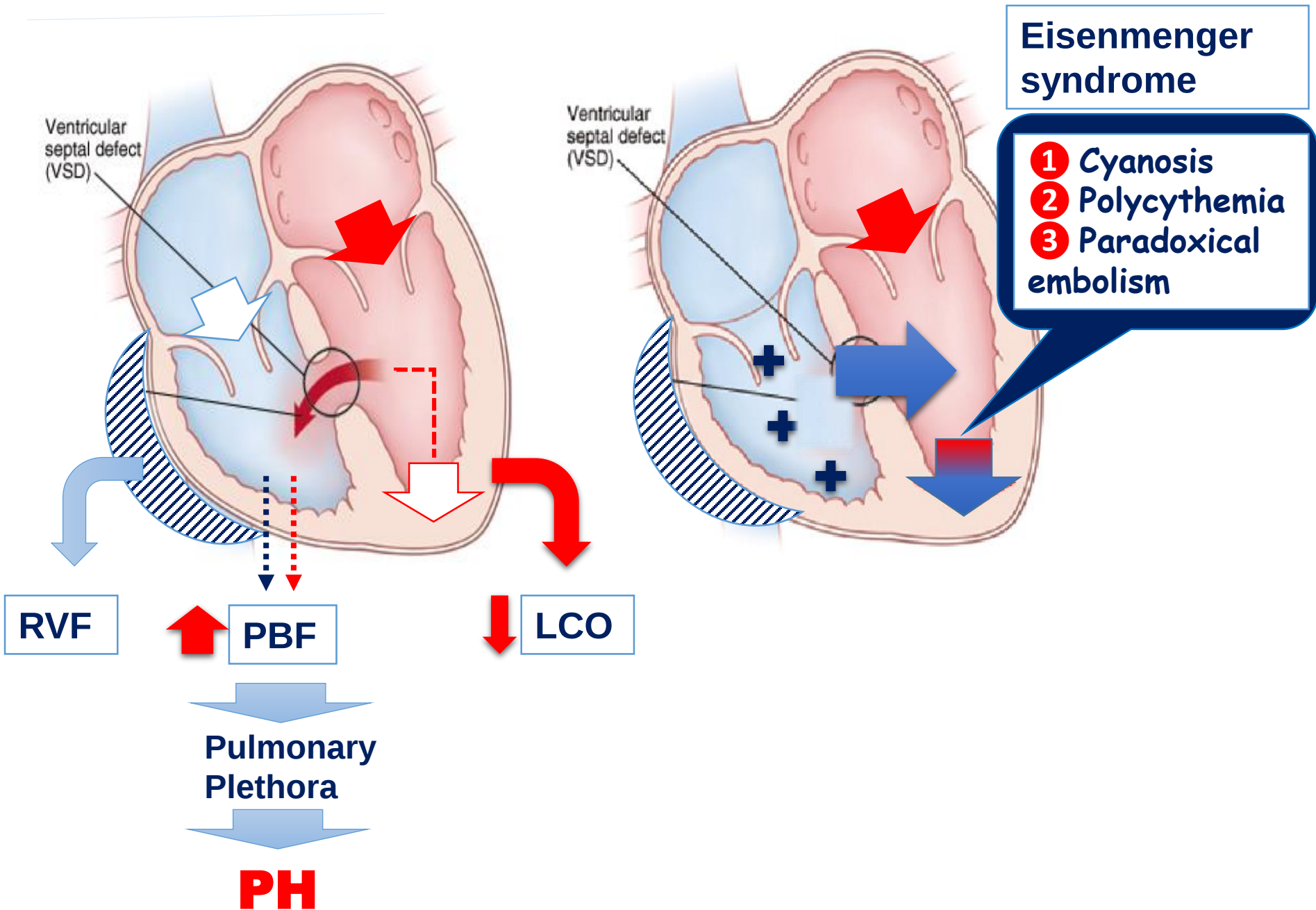
VSD is a defect in the interventricular septum



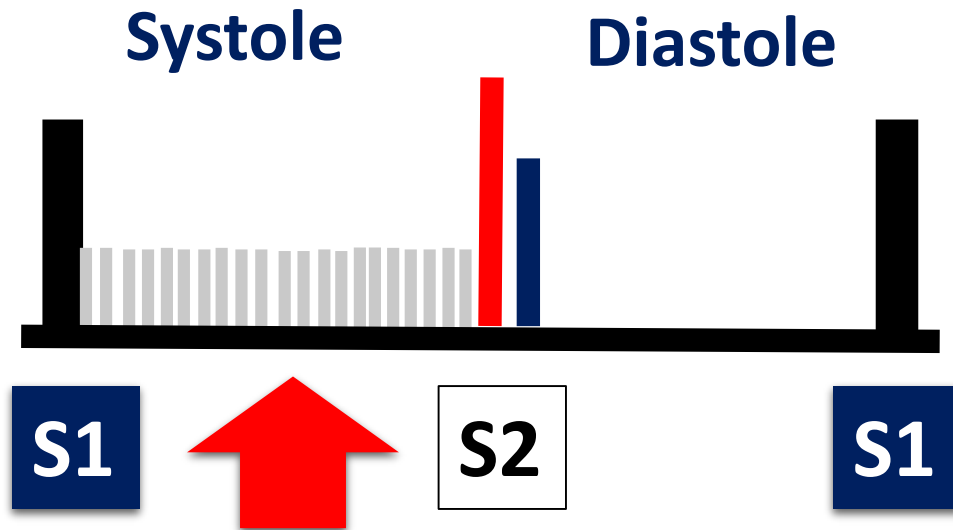
Aetiology

Most likely Congenital or Acquired e.g. in AMI

Clinical Picture



Auscultation



PSM

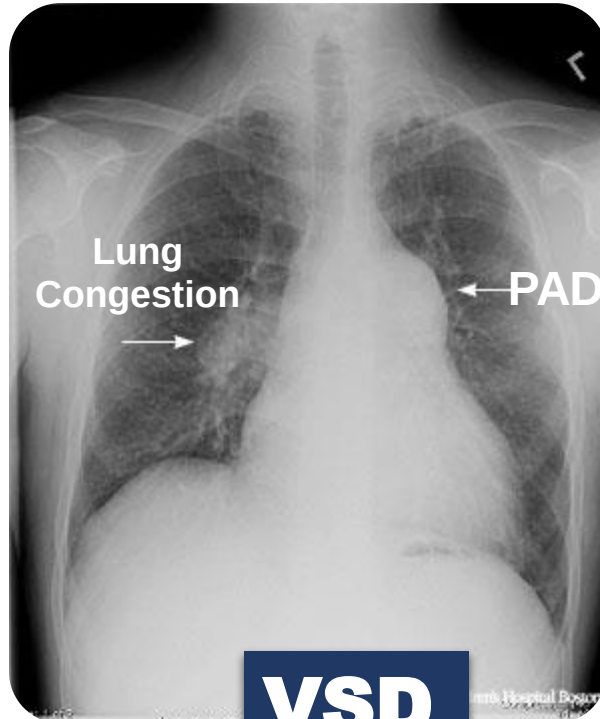
- 1- Harsh Murmur
- 2- Associated with a Thrill
- 3- Over 3rd and 4th third and fourth ICS on left sternal border

Special Investigations

- ① RVH is seen on ECG
- ② PAD and increased Lung vascularity on Chest X-Ray



Normal



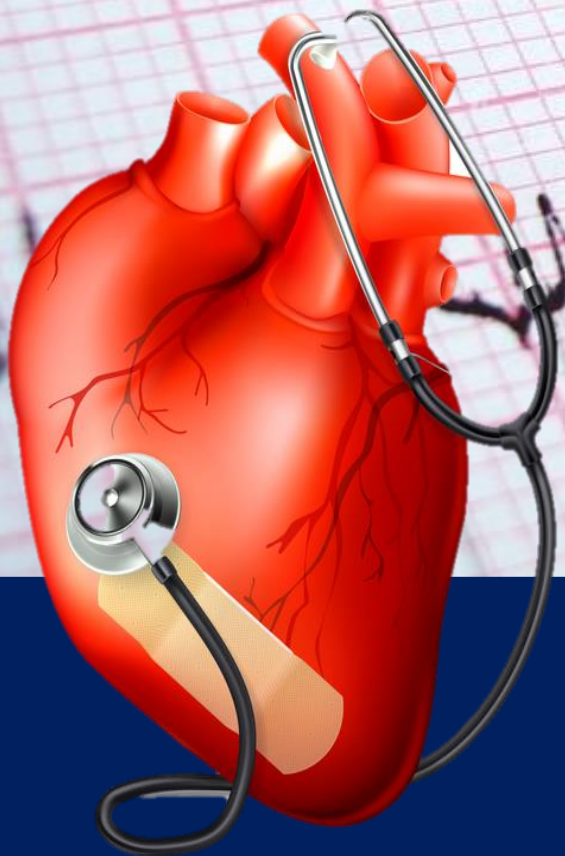
VSD

- ③ Echo-Doppler-Doppler-MRI- and Catheter confirms the Diagnosis

Treatment

Surgery **Plus Prophylaxis against SBE**

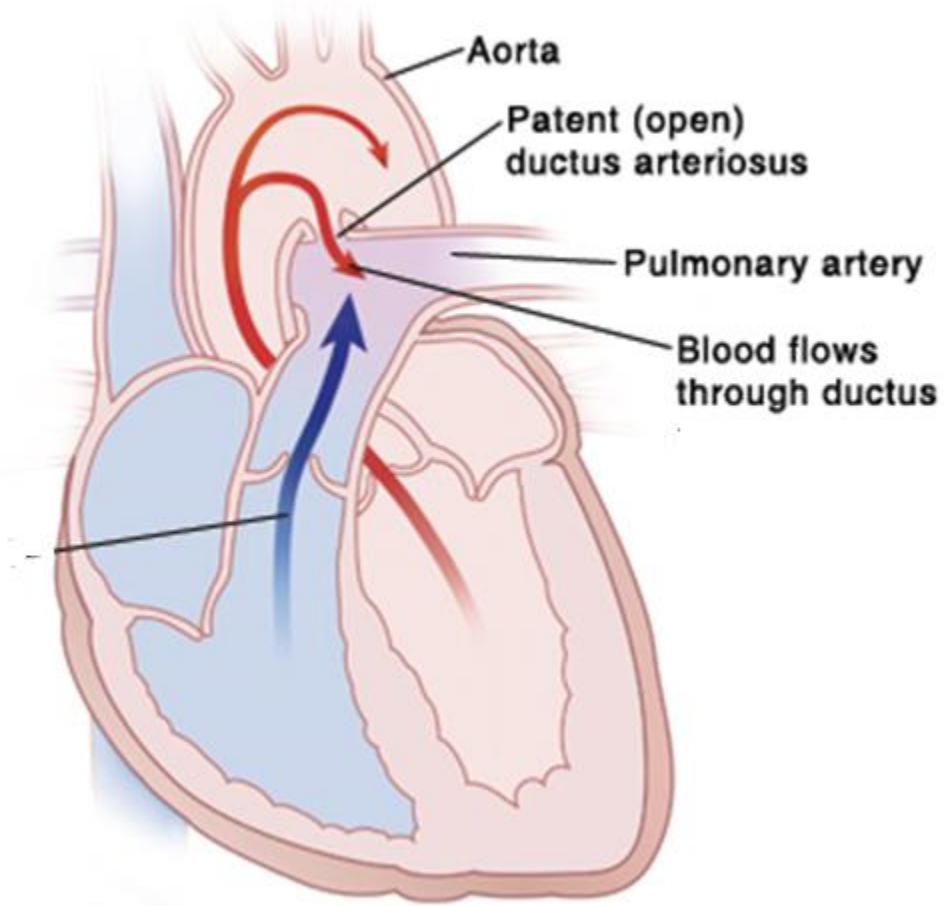
Cardiology



PDA

Definition

A shunt that connects the Aorta with the PA directly.

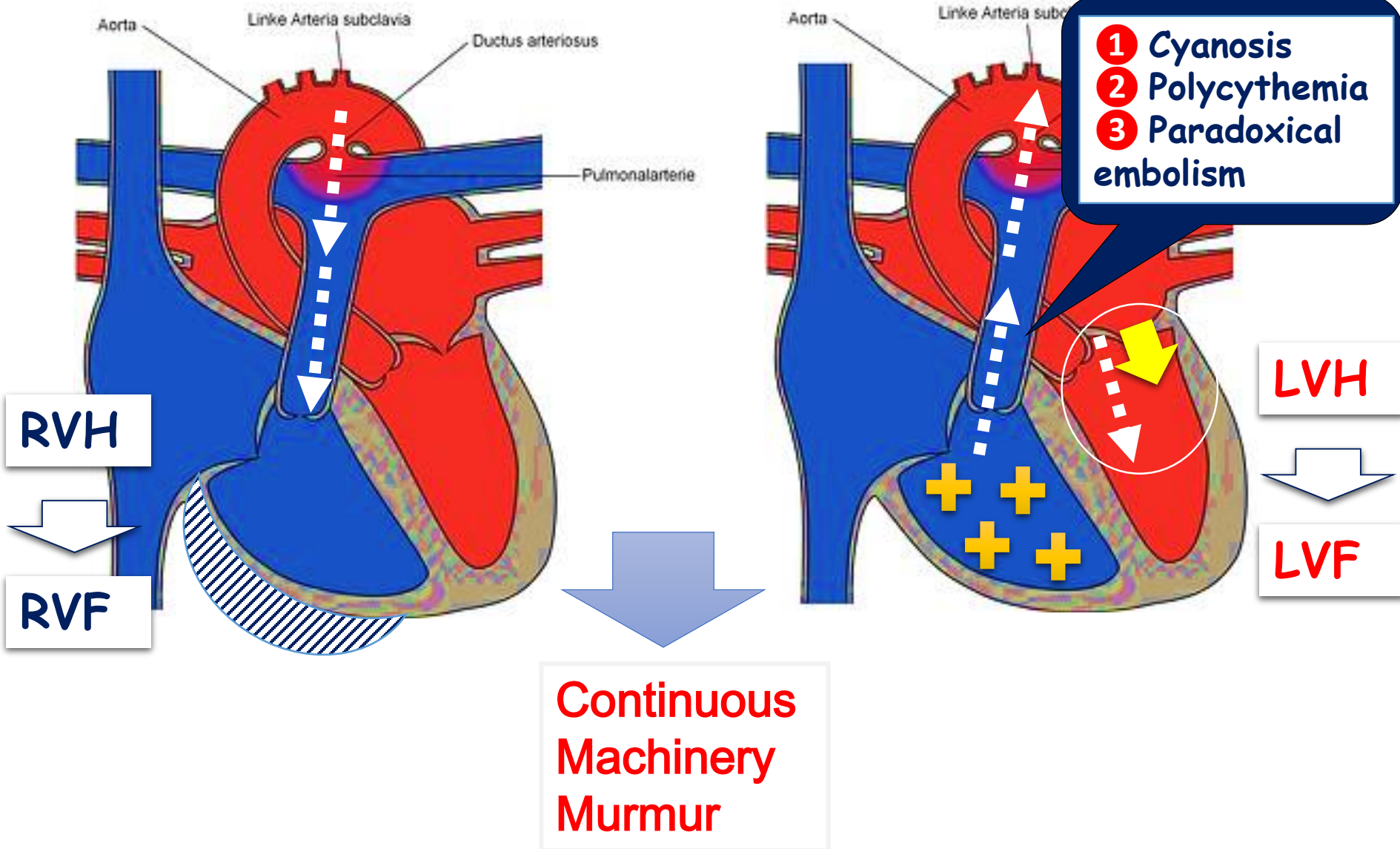


Aetiology

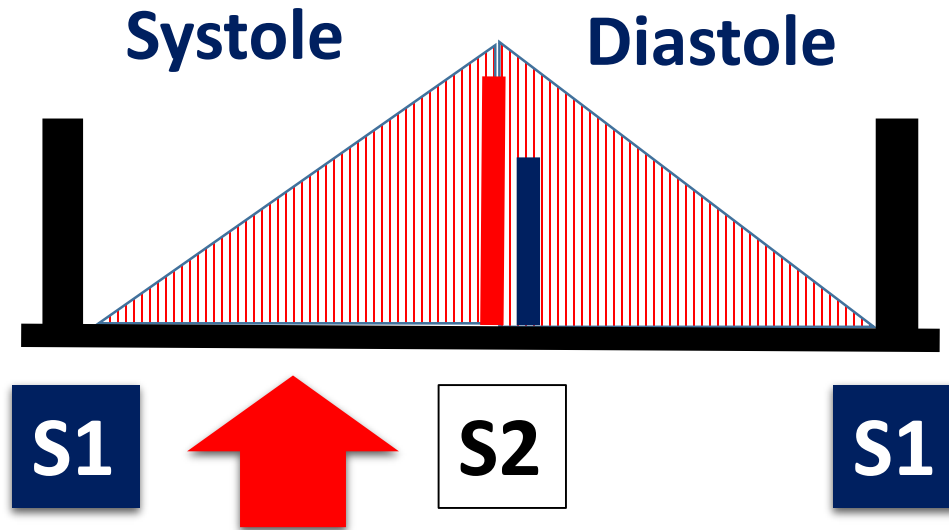
Congenital

Clinical Picture

Eisenmenger syndrome



Auscultation



**Continuous
Machinery Murmur**

Note: This Murmur will **diminish** when heart failure develops.

Special Investigations

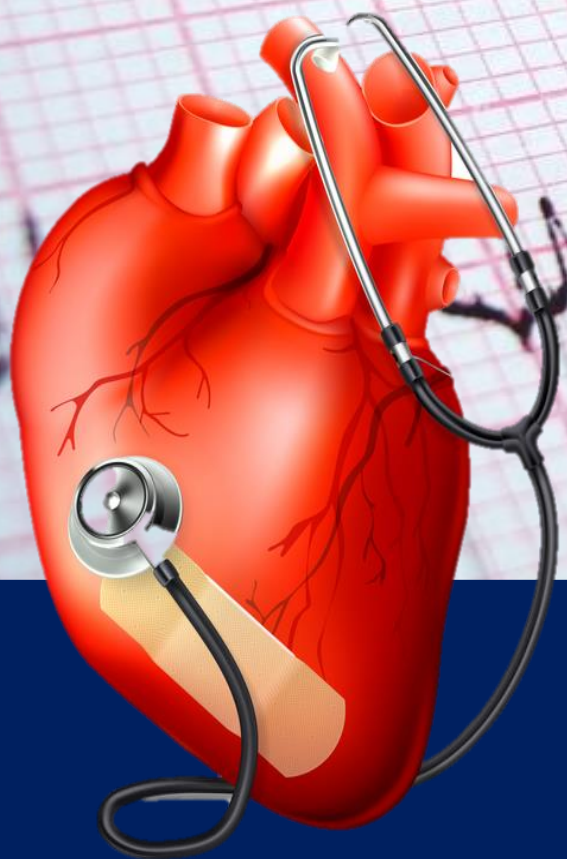
**Echo-Doppler--MRI- and
Catheter confirms the
Diagnosis**

Treatment

- ① Surgical Correction
- ② **Indomethacin** Immediately after Birth
(by its Anti PG effect it enhances the closure of the defect)
- ③ Prophylaxis against SBE



Cardiology



Pulmonary Stenosis

تحذير هام

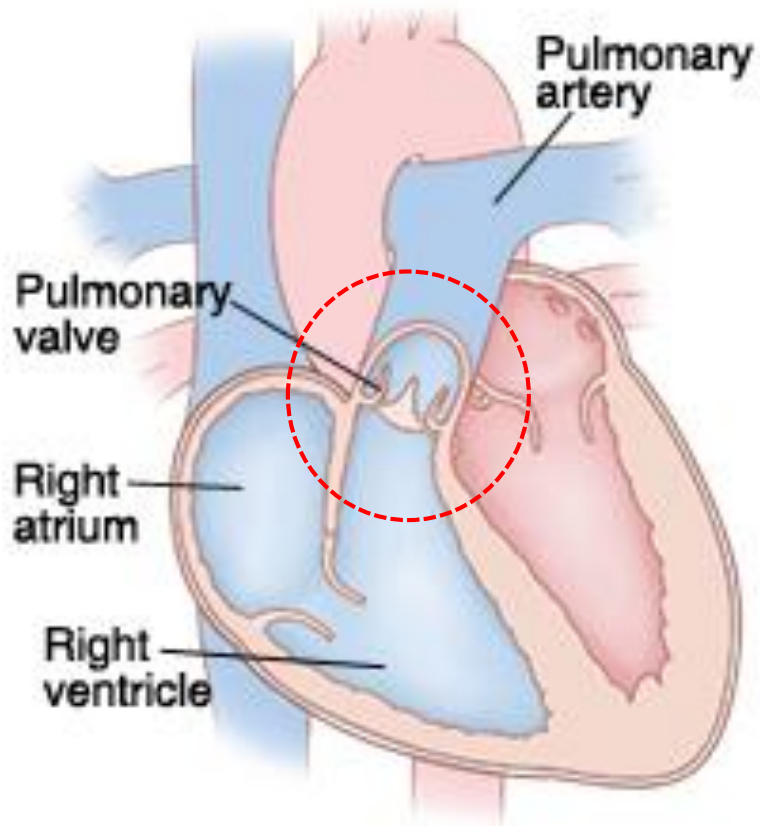
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Definition

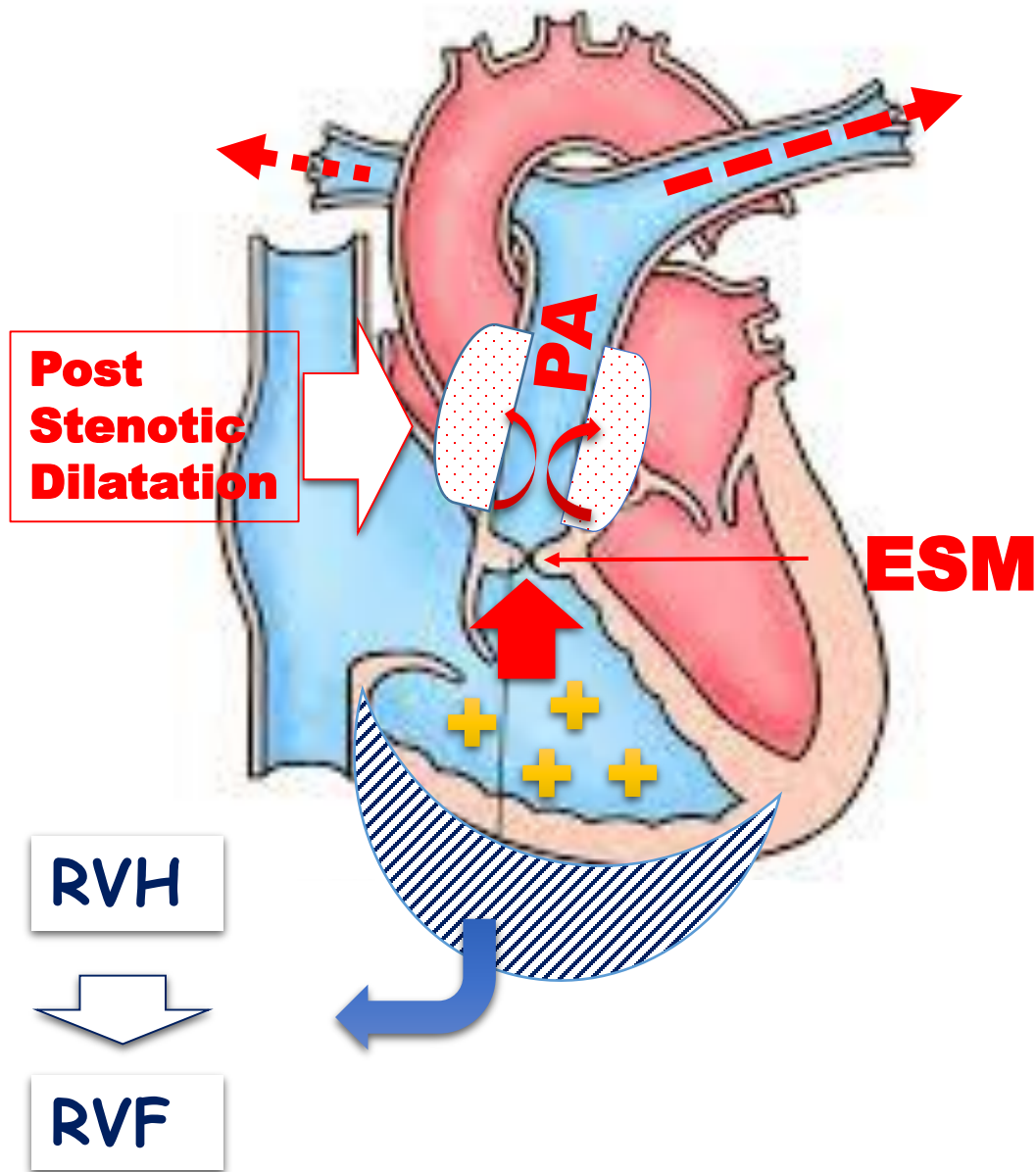
Stenosis of the Pulmonary Valve



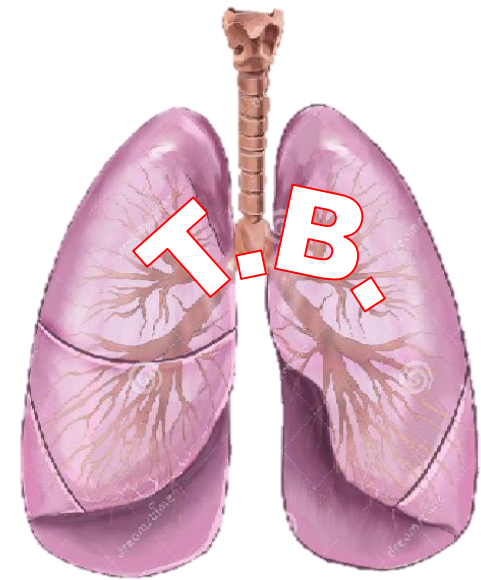
Aetiology

Mostly Congenital

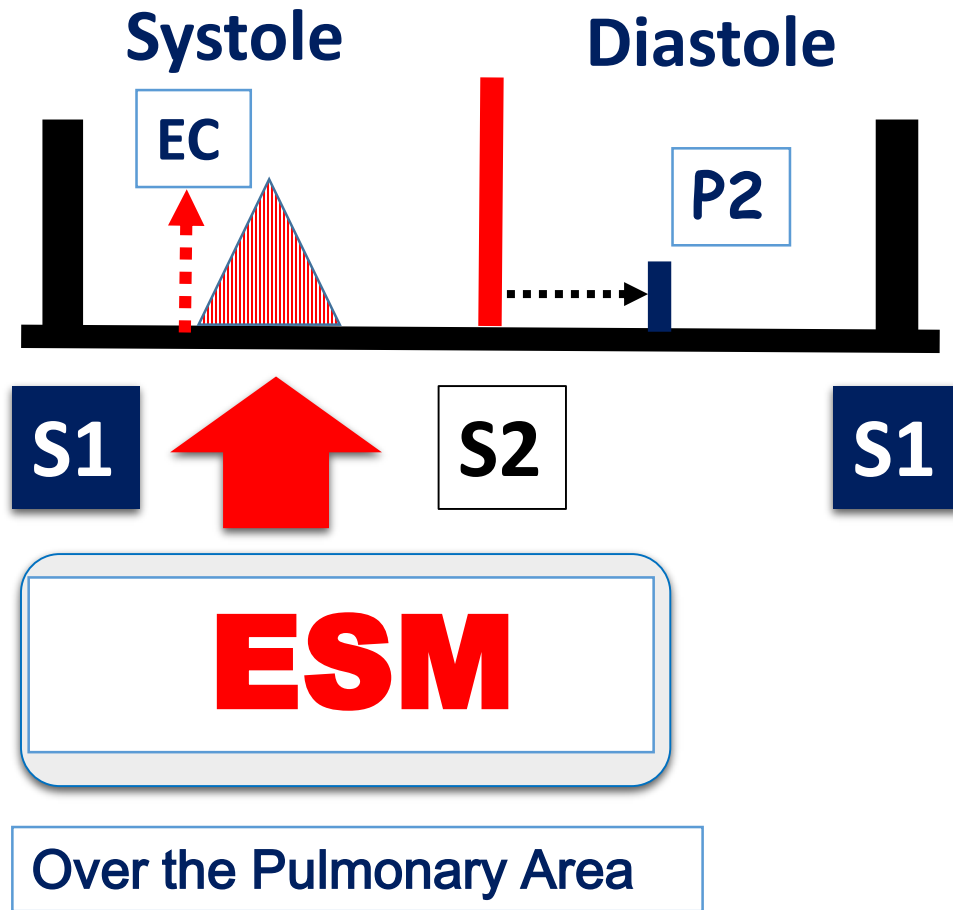
Clinical Picture



Pulmonary Oligemia



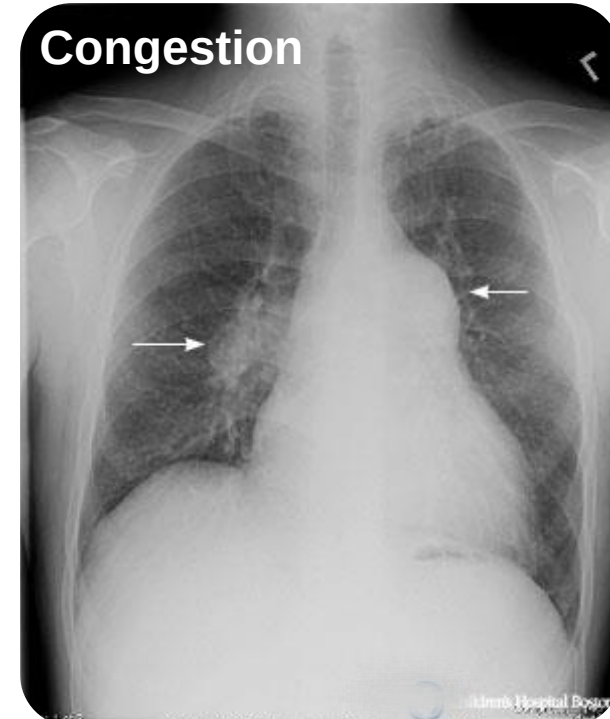
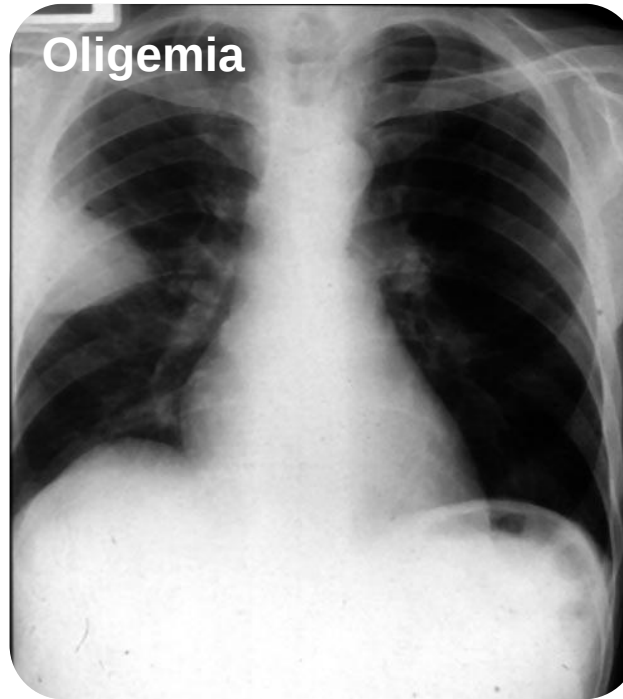
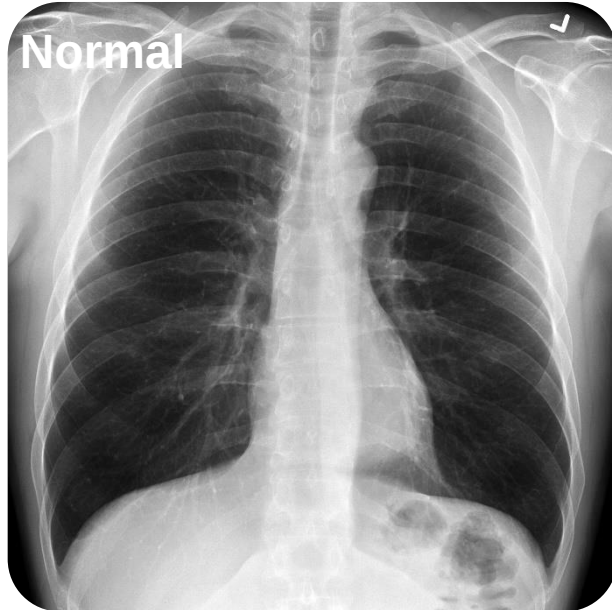
Auscultation



Special Investigations

① ECG: RVH

② Lung Oligemia on Chest X-Ray

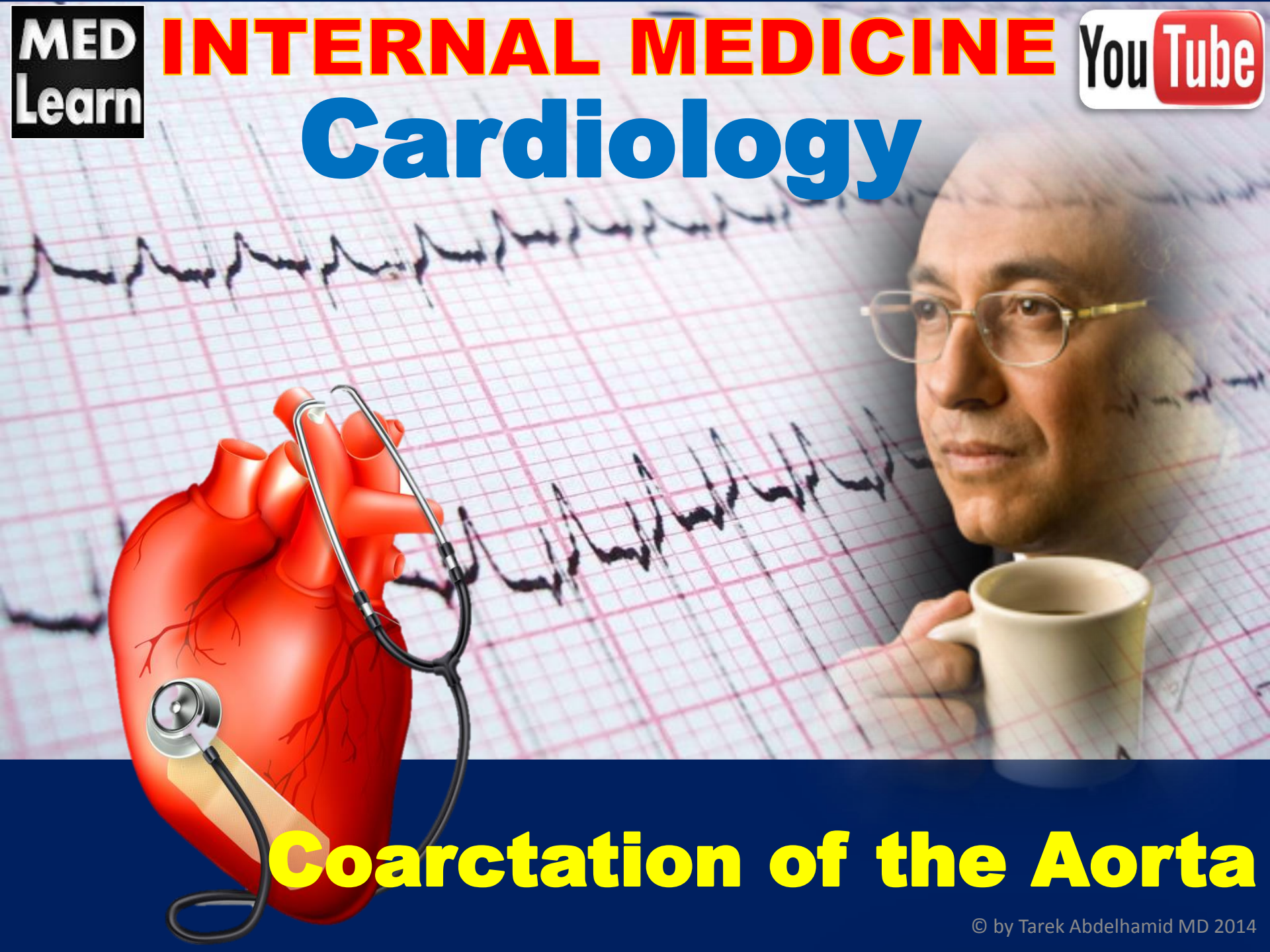


③ Echo-Doppler-Doppler-MRI-
confirms the Diagnosis

Treatment

Surgical

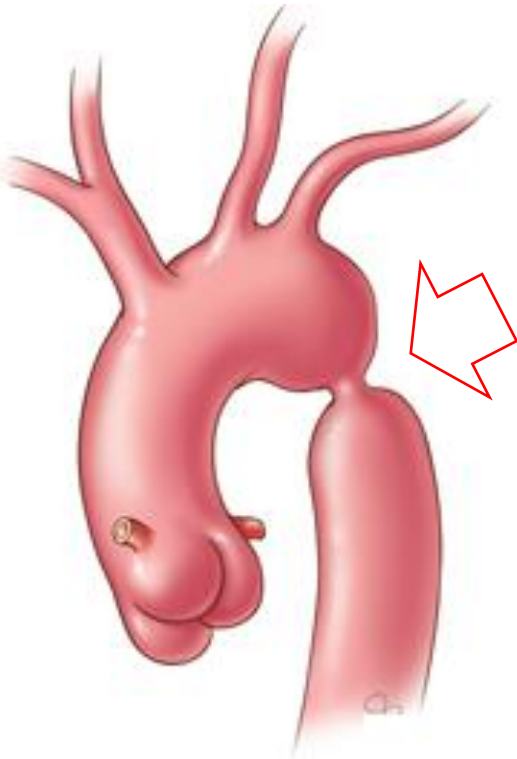
Cardiology



Coarctation of the Aorta

Definition

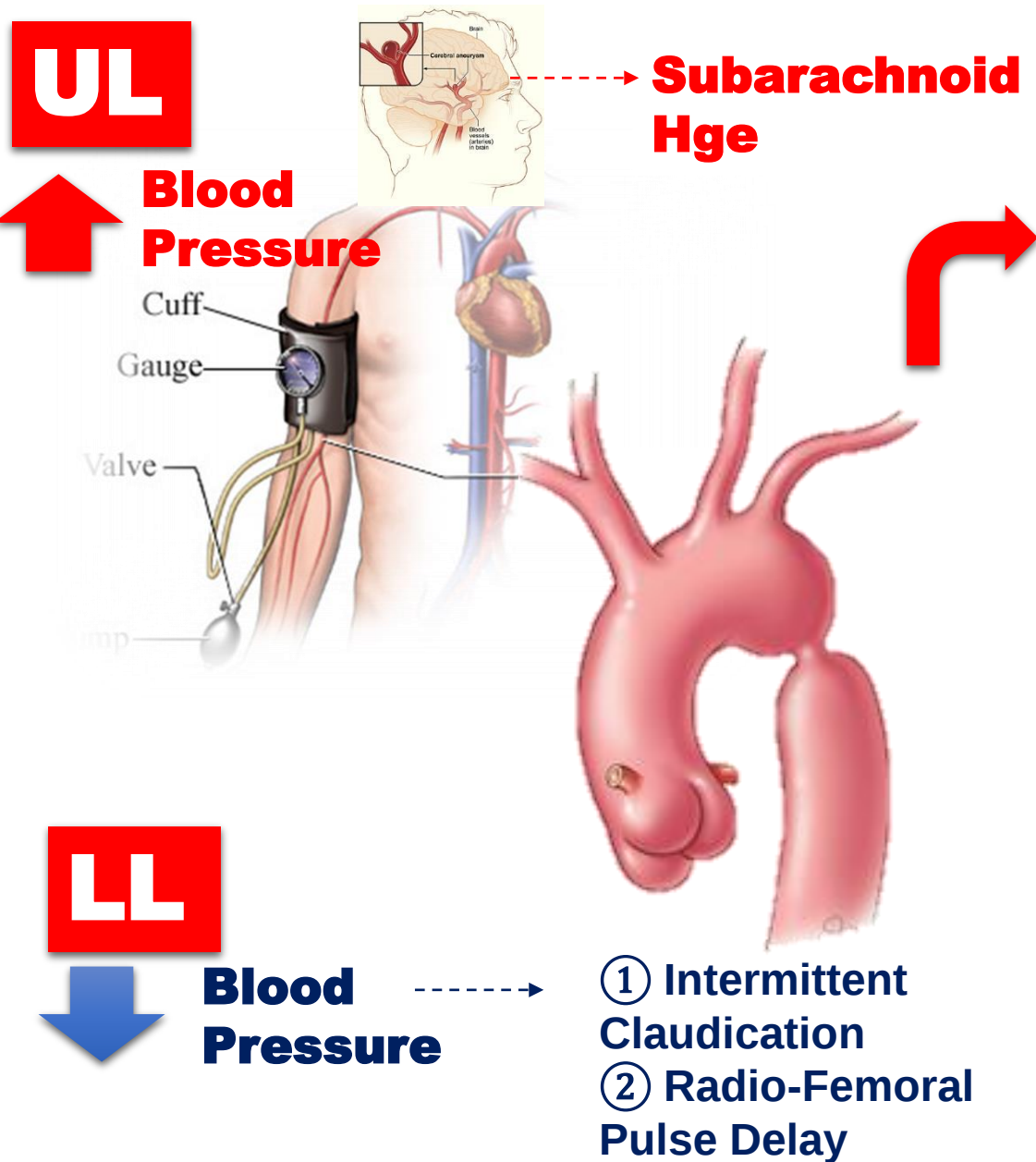
Narrowing of a segment of the Aorta (usually thoracic aorta)



Aetiology

Congenital

Clinical Picture

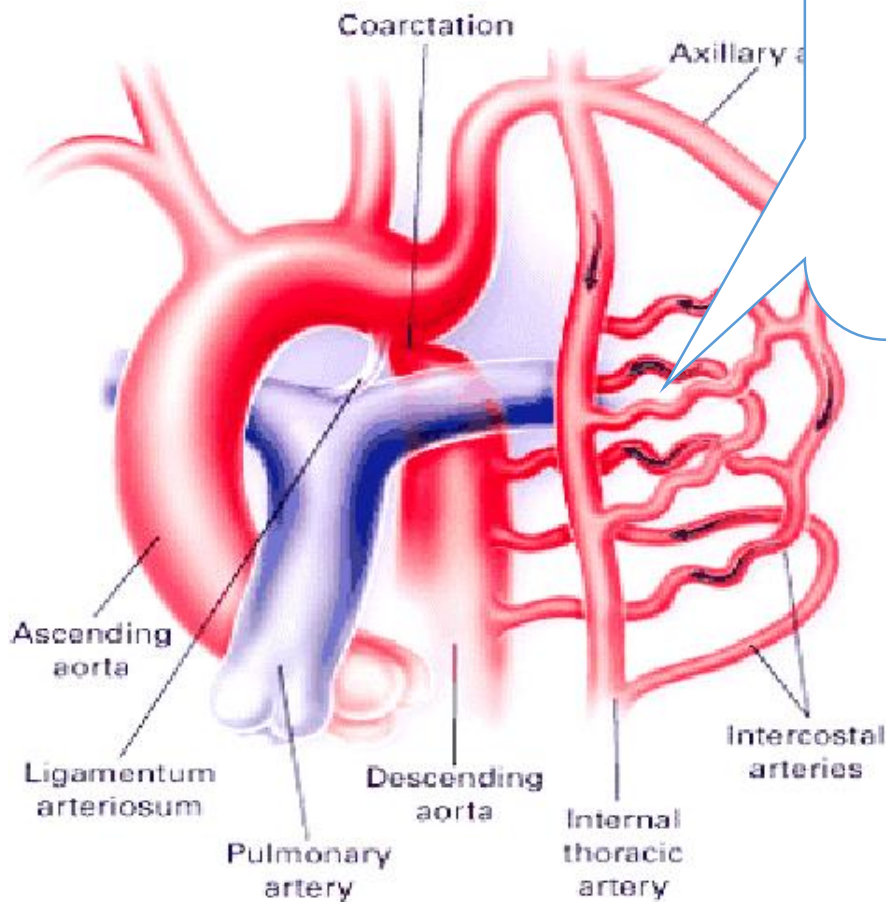


Before opening the Collaterals!

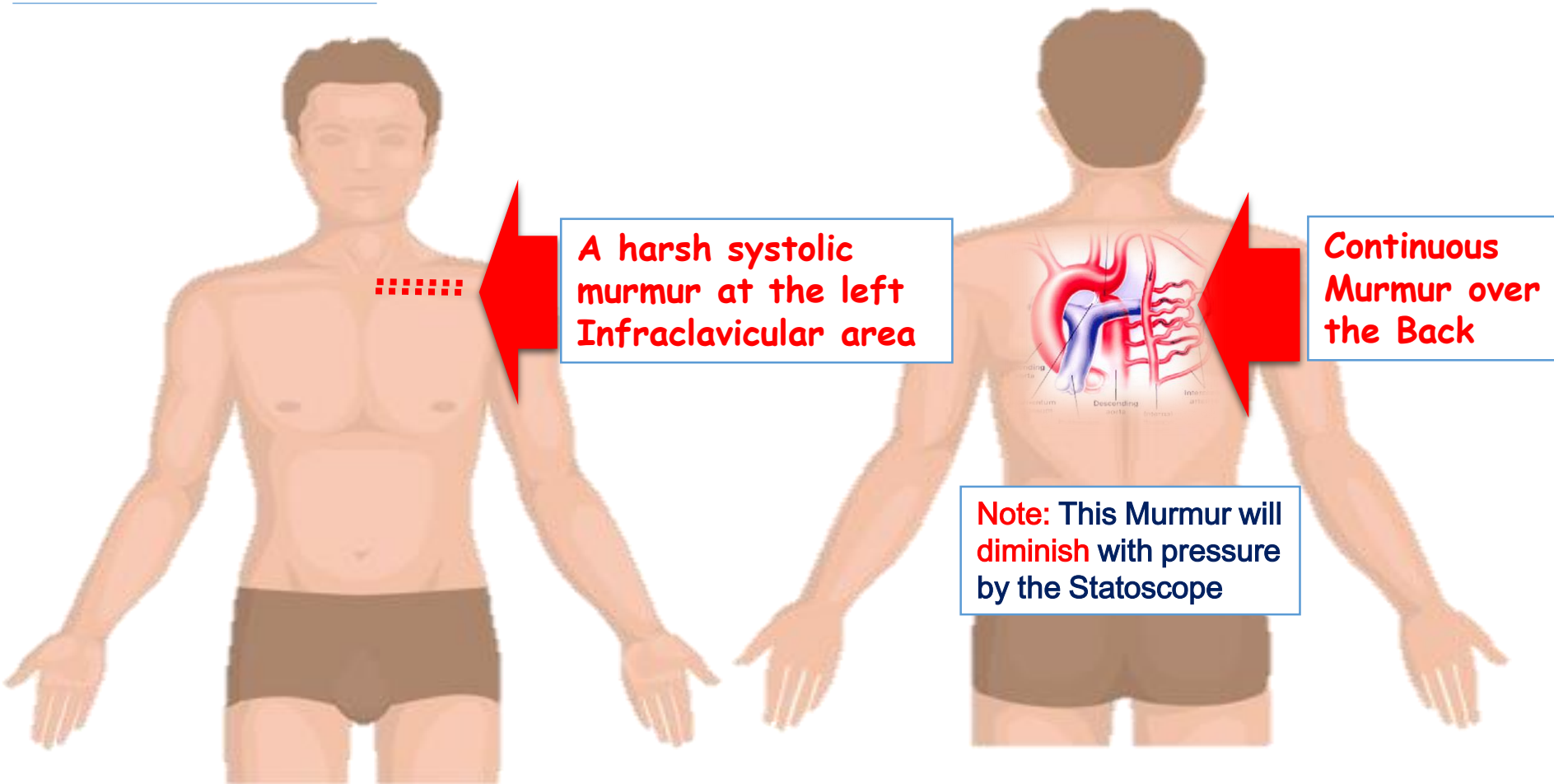
Clinical Picture

Collaterals

- 1 Continuous murmur over the back
- 2 Visible and palpable pulsations in the Inter Scapular Region
- 3 Left Shoulder pain
- 4 Erosion on the Lower surface of the ribs (X-Ray)



Auscultation



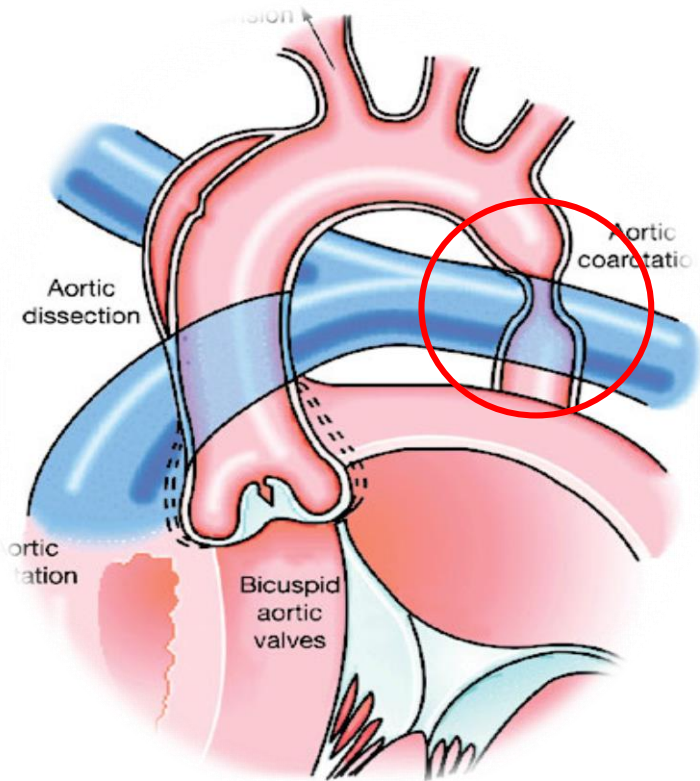
Special Investigations

Echo-Doppler-Doppler-MRI- and Catheter confirms the Diagnosis



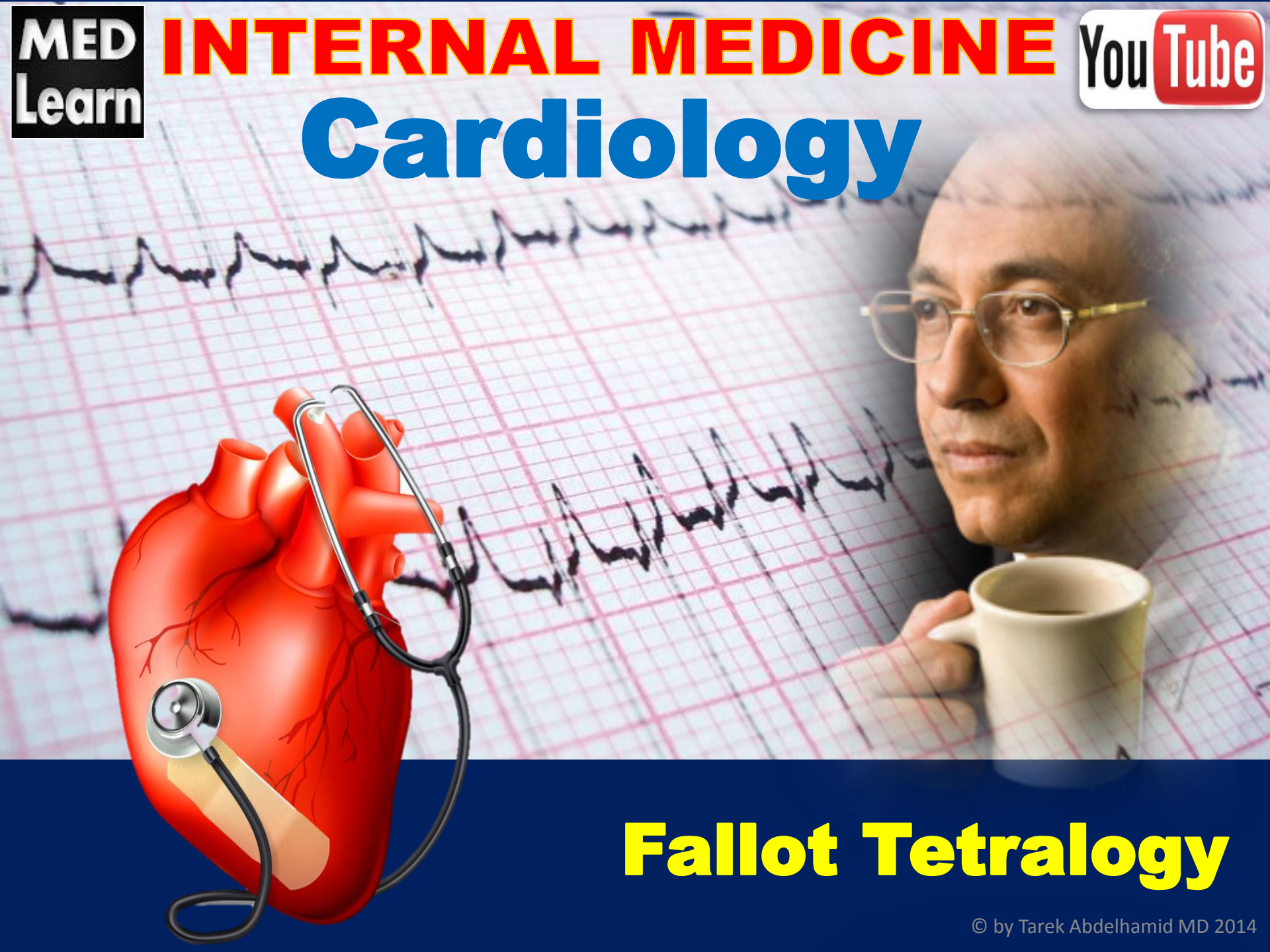
Treatment

Essentially Surgical



Note: PGE2 could be used to open the collaterals and decrease the CHF in infants

Cardiology

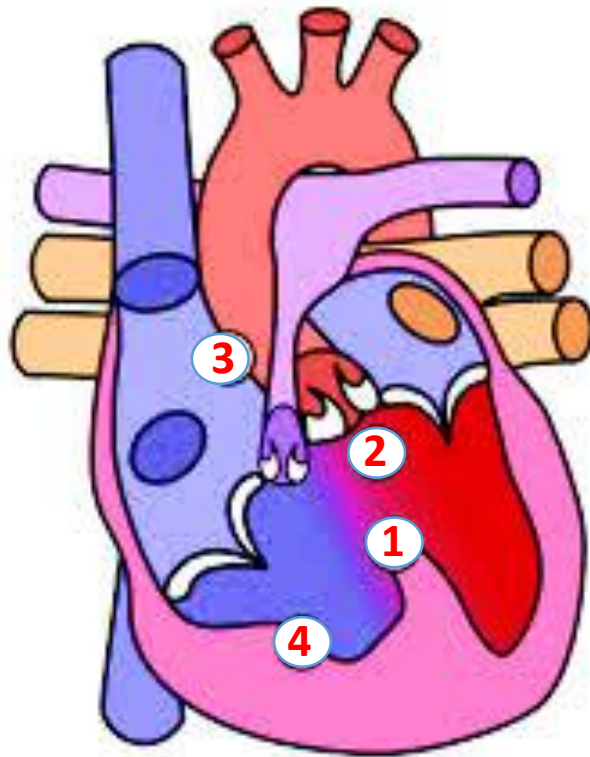


Fallot Tetralogy

Definition

Fallot Tetralogy is a congenital heart condition characterized by the presence of

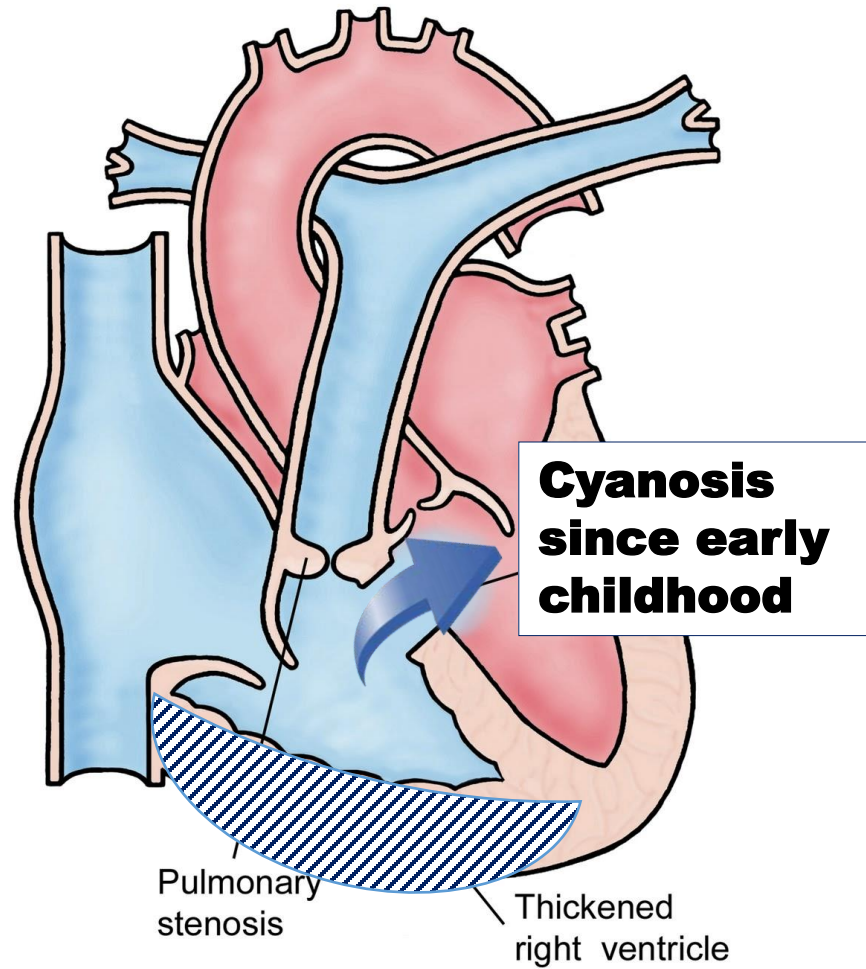
- ① VSD
- ② Overriding of the Aorta
- ③ Pulmonary artery Stenosis
- ④ 'Mild' RVH



Aetiology

Most likely Congenital

Clinical Picture

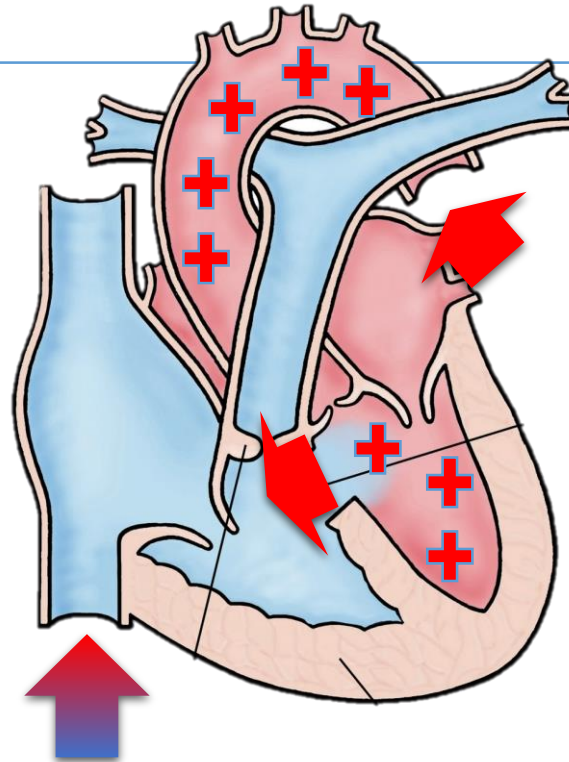
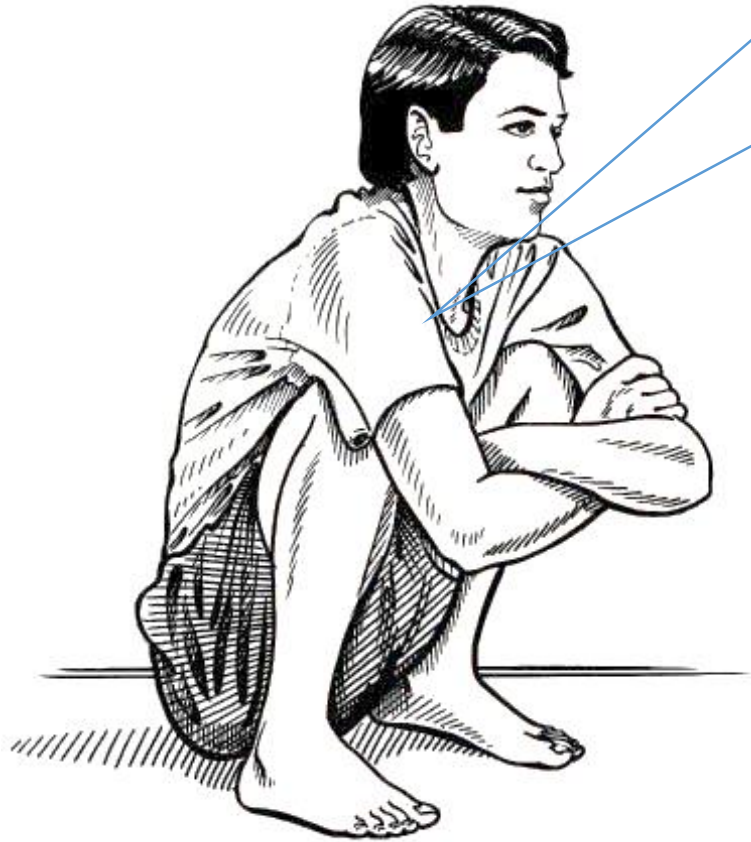


Cyanotic Spills



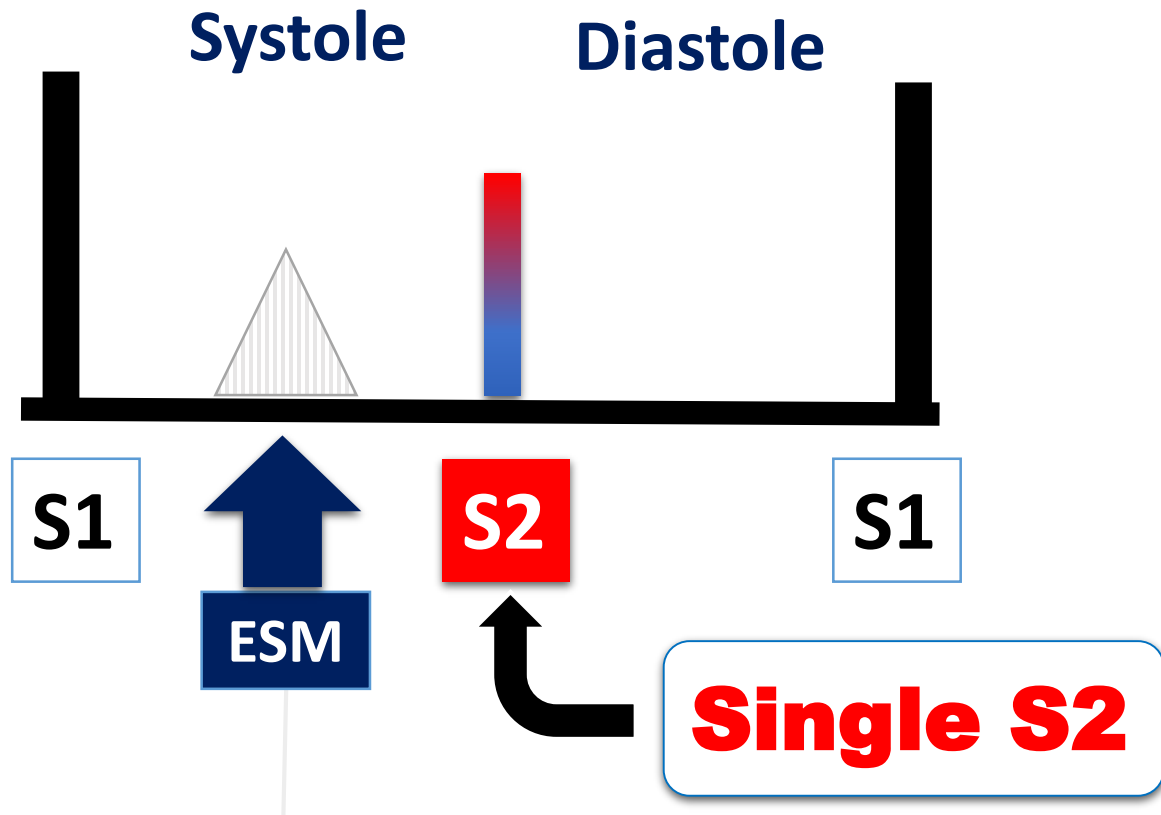
Why?

- 1 Increase VR from the more oxygenated splanchnic circulation
- 2 Increase resistance of the systemic arterial system (due to kink of the femoral artery) which forces more blood to be directed to the pulmonary circulation



Cyanotic attacks are typically relived by Squatting

Auscultation



Note: this murmur typically decreases during the Cyanotic Spills due to decrease pulmonary blood flow.

Special Investigations

**Echo-Doppler-Doppler-MRI-
and Catheter confirms the
Diagnosis**

Treatment

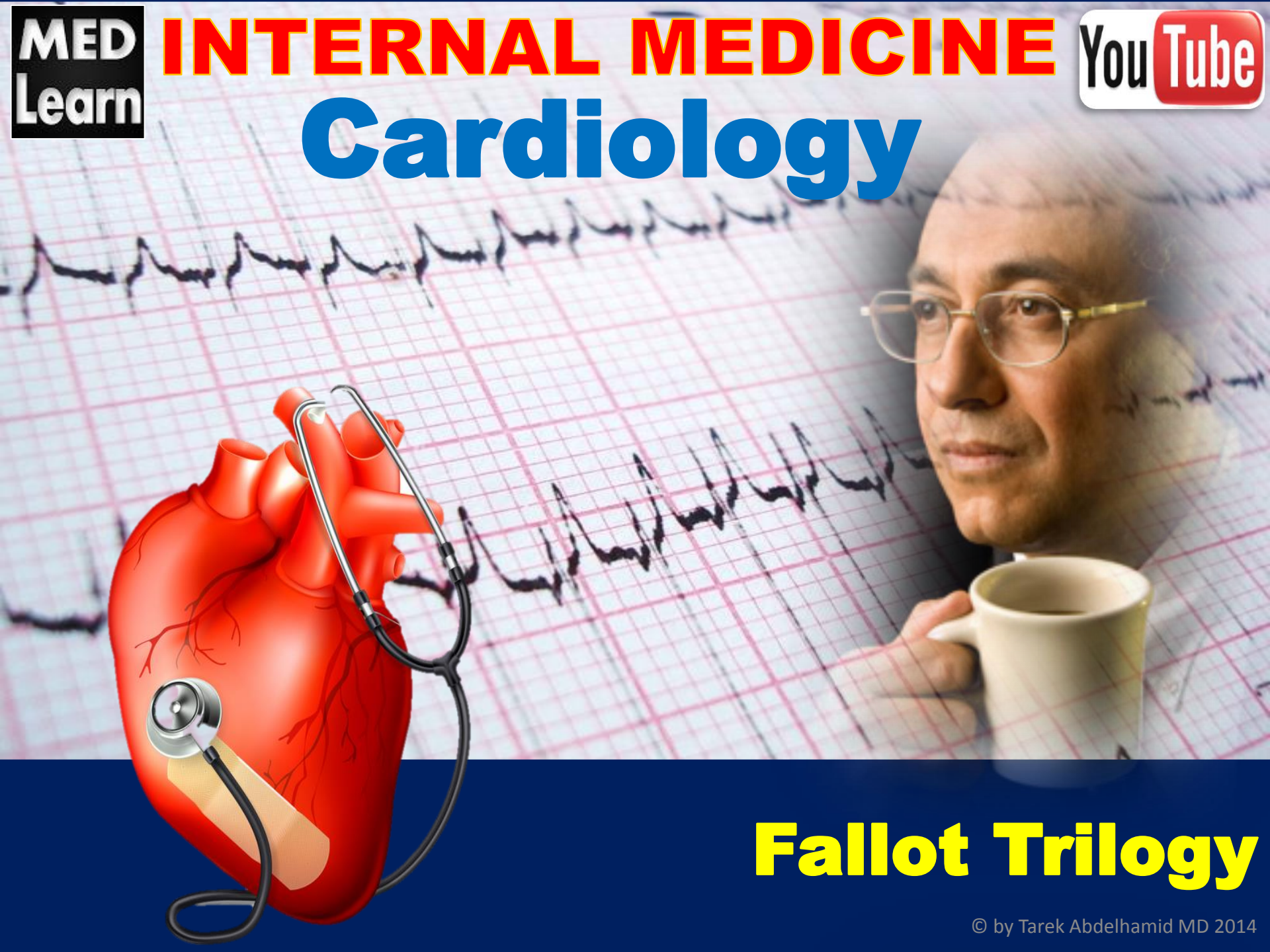
Surgery

Prophylaxis against SBE

Treatment of Cyanotic Spills

- 1 Squatting
- 2 O2
- 3 Morphia
- 4 beta blockers

Cardiology

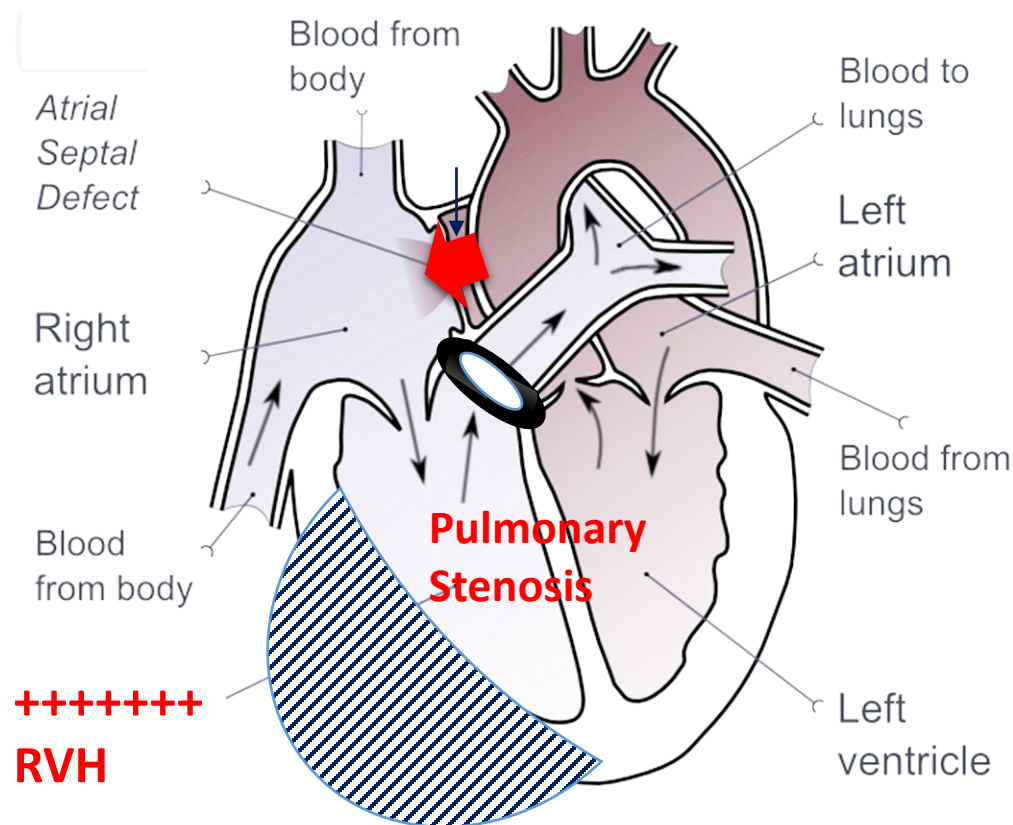


Fallot Trilogi

Definition

A cardiac condition characterized by the presence of:

- 1 Pulmonary Stenosis (**Valvular**)
- 2 ASD
- 3 +++ RVH



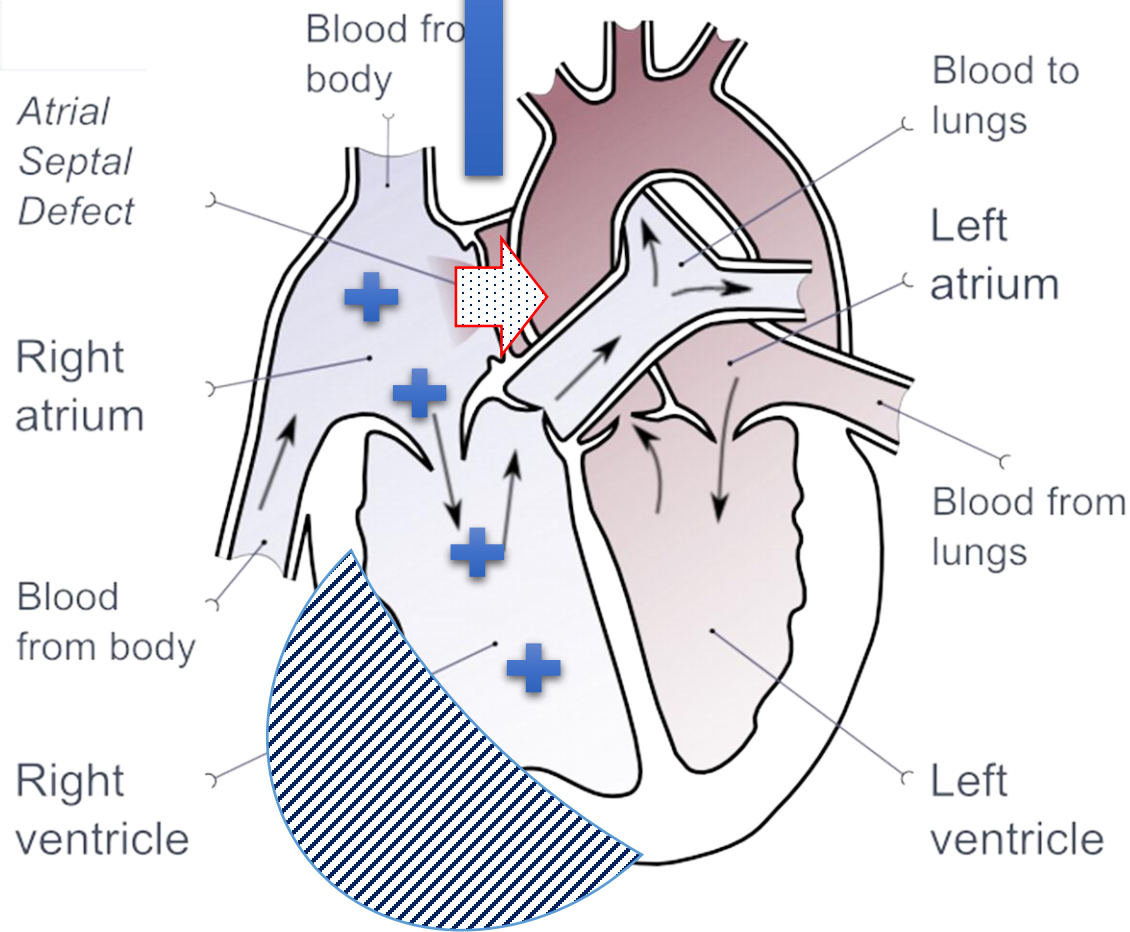
Aetiology

Most likely Congenital

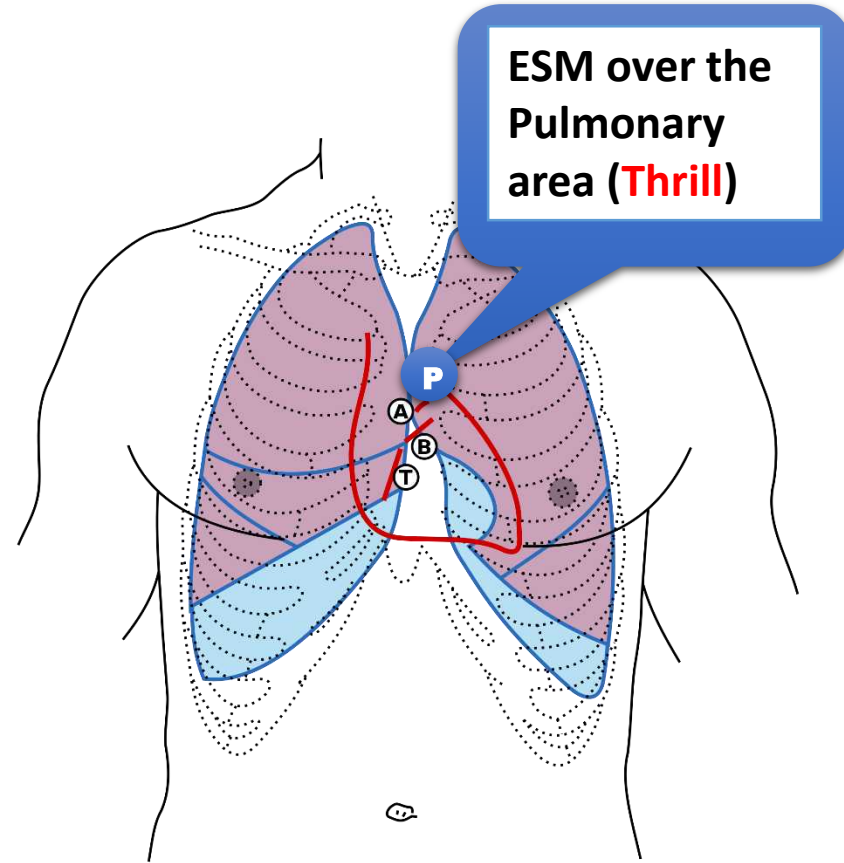
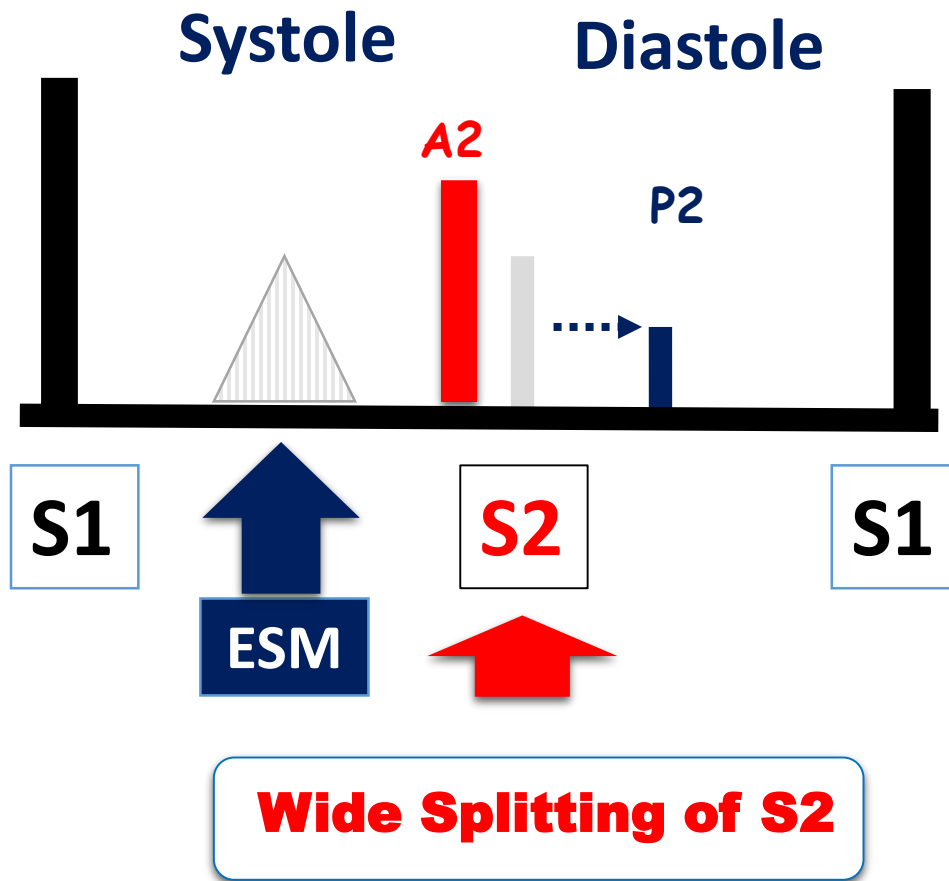
Clinical Picture

**Cyanosis
(Late)**

**NO effect for
squatting**



Auscultation



Special Investigations

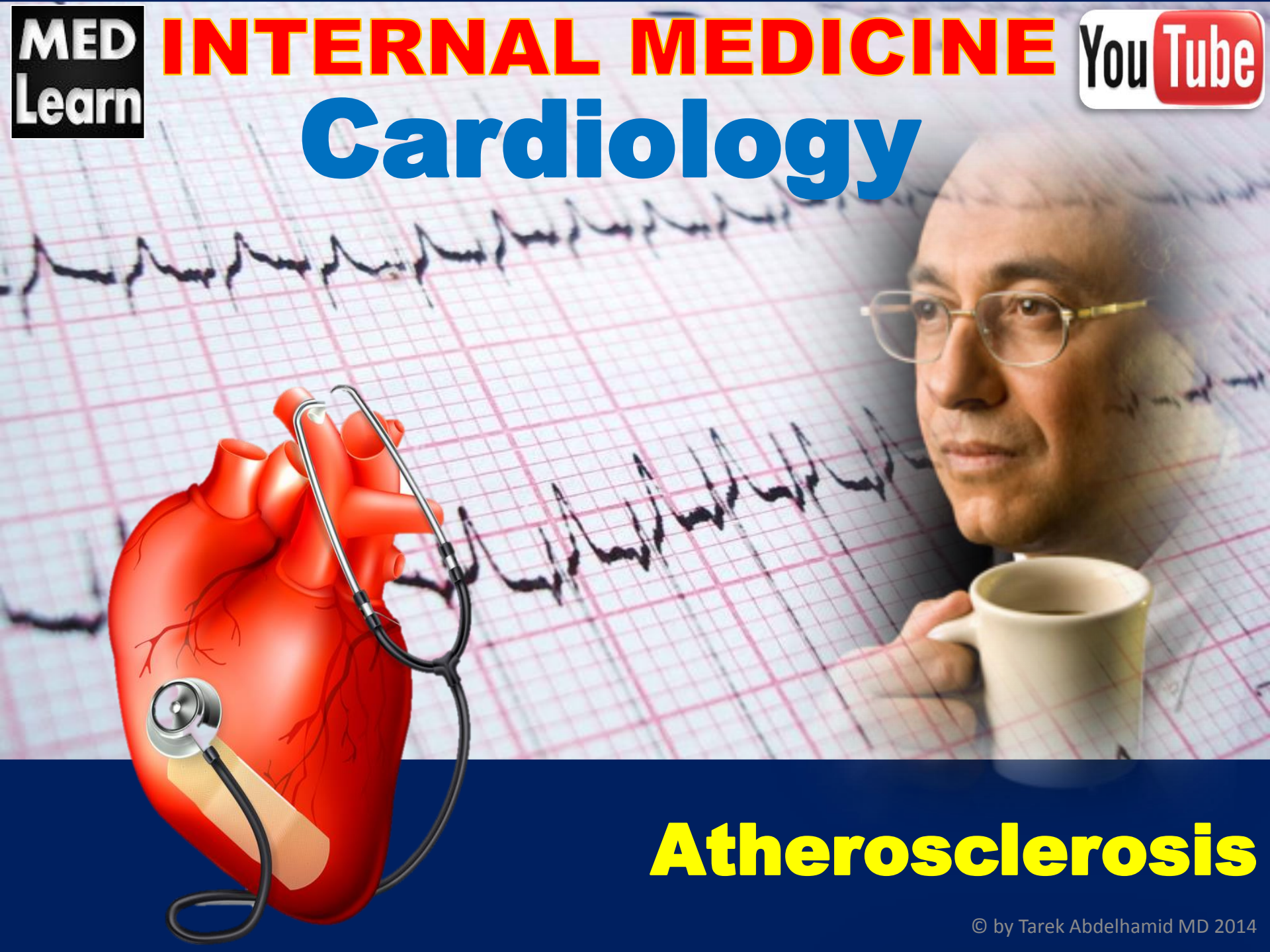
Echo-Doppler-Doppler-MRI- and Catheter confirms the Diagnosis

Treatment

Surgery

Prophylaxis against SBE

Cardiology

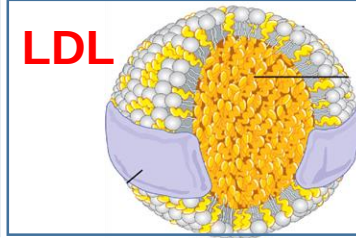
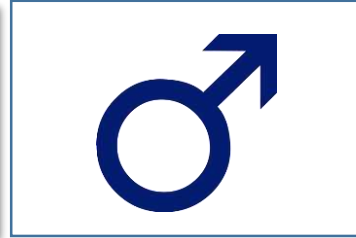


Atherosclerosis

Definition

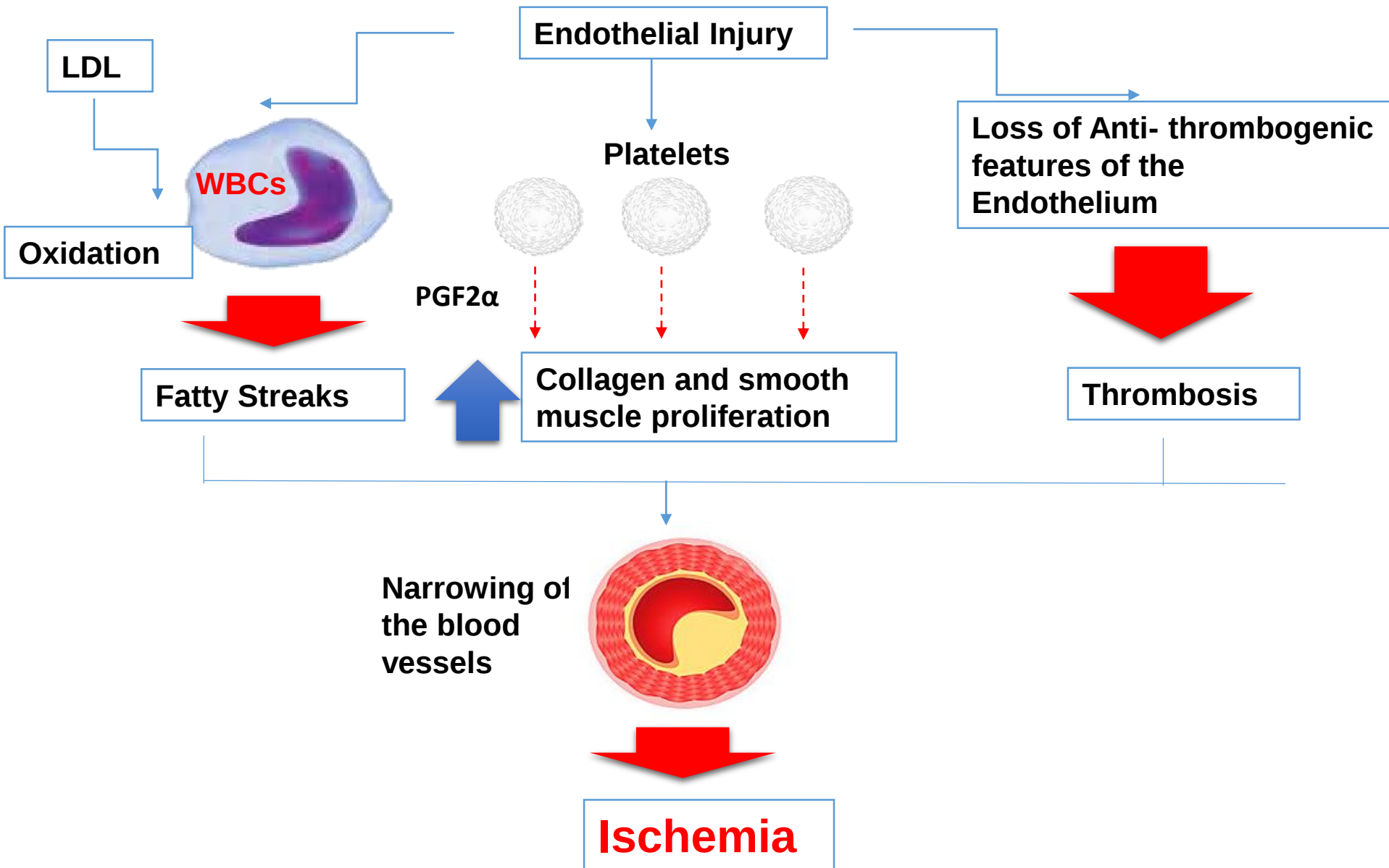
A chronic degenerative process of the wall of blood vessels (usually the elastic arteries).

Predisposing Factors



- 1- Cigarette Smoking
- 2- Diabetes Mellitus
- 3- Hyperlipidemias ( LDL/HDL)
- 4- Hypertension
- 5- Male Sex
- 6- ?! Genetic
- 7- Age
- 8- Others: +++ Homocysteine-  Lipoprotein A- Stress- Obesity- Lack of exercise

Pathogenesis



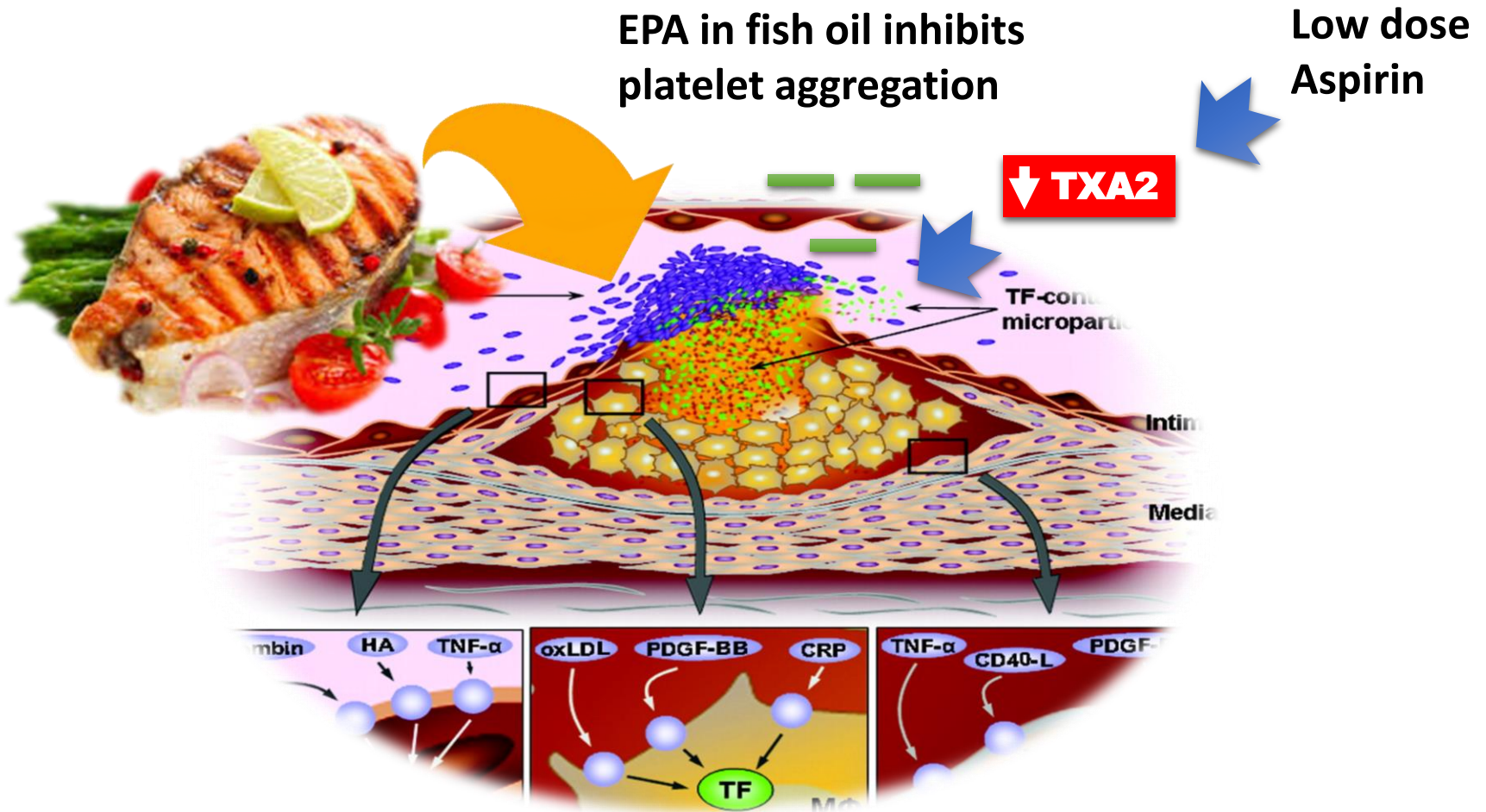
Clinical Picture

The affected vessels become harder, thicker, and narrower thus causes ischemia of the organs or tissues supplied by such vessels. This may lead to:

- ① IHD
- ② Stroke
- ③ Ischemia of the Lower Limb

Treatment

① Aspirin (**Low dose**) ② Fish ③ Vitamin E (↓ **LDL Oxidation**) ④
Treatment of Predisposing Factors ⑤ **STOP** Smoking



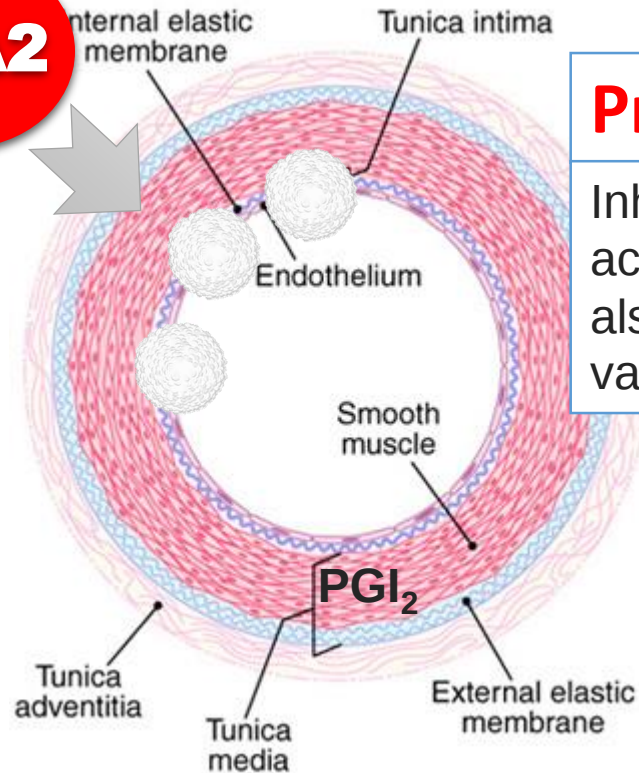
**Low
dose
Aspirin**

**High
dose
Aspirin**

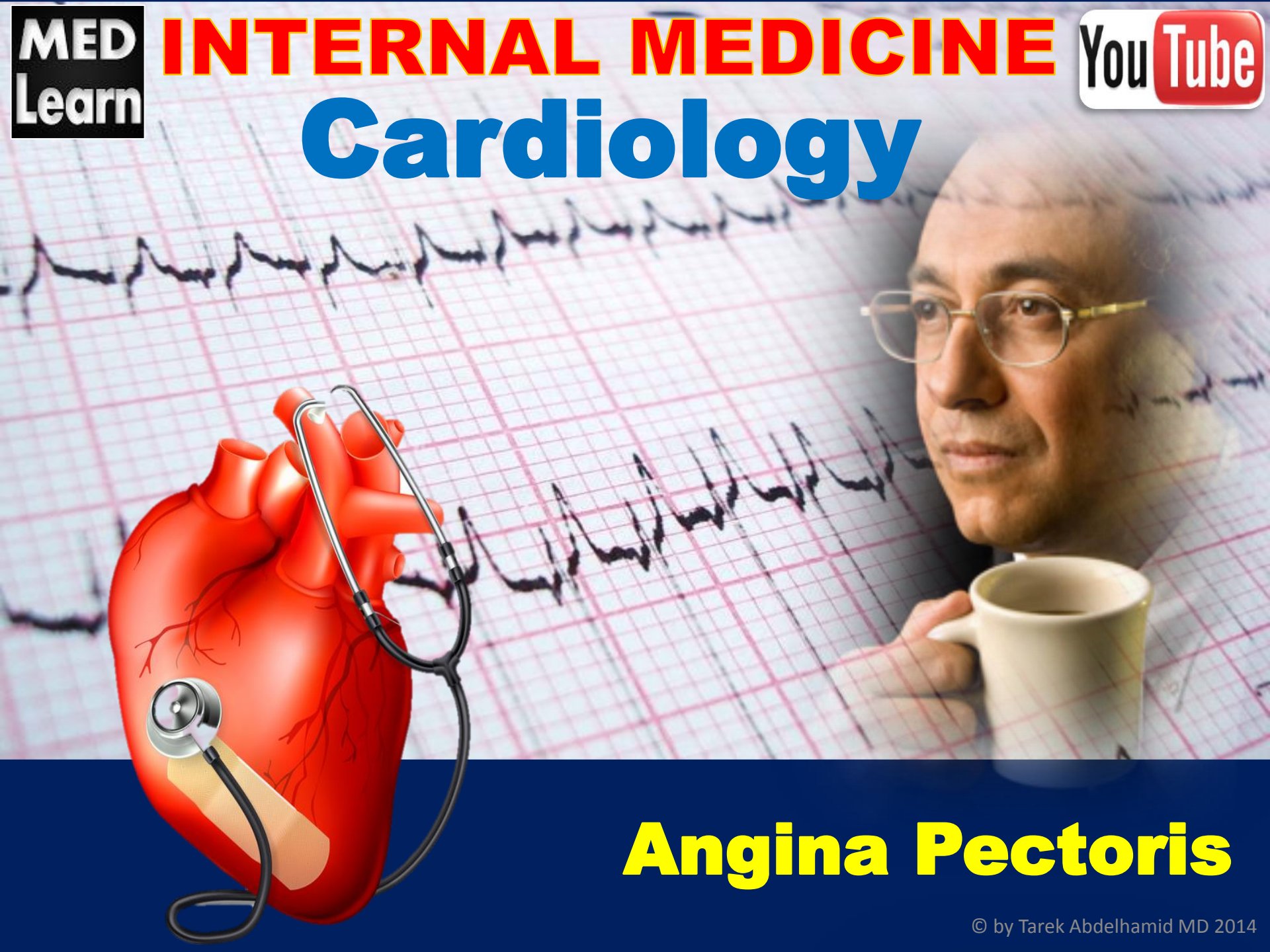
TXA₂

Prostacyclin

Inhibits platelet activation and is also an effective vasodilator.



Cardiology



Angina Pectoris

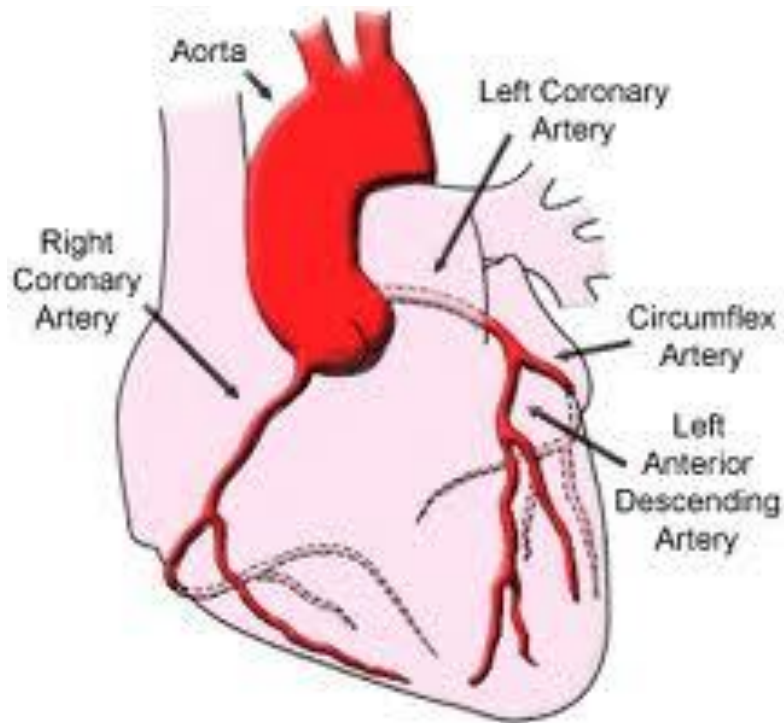
Definition

Recurrent attacks of Chest Pain caused by myocardial ischemia.

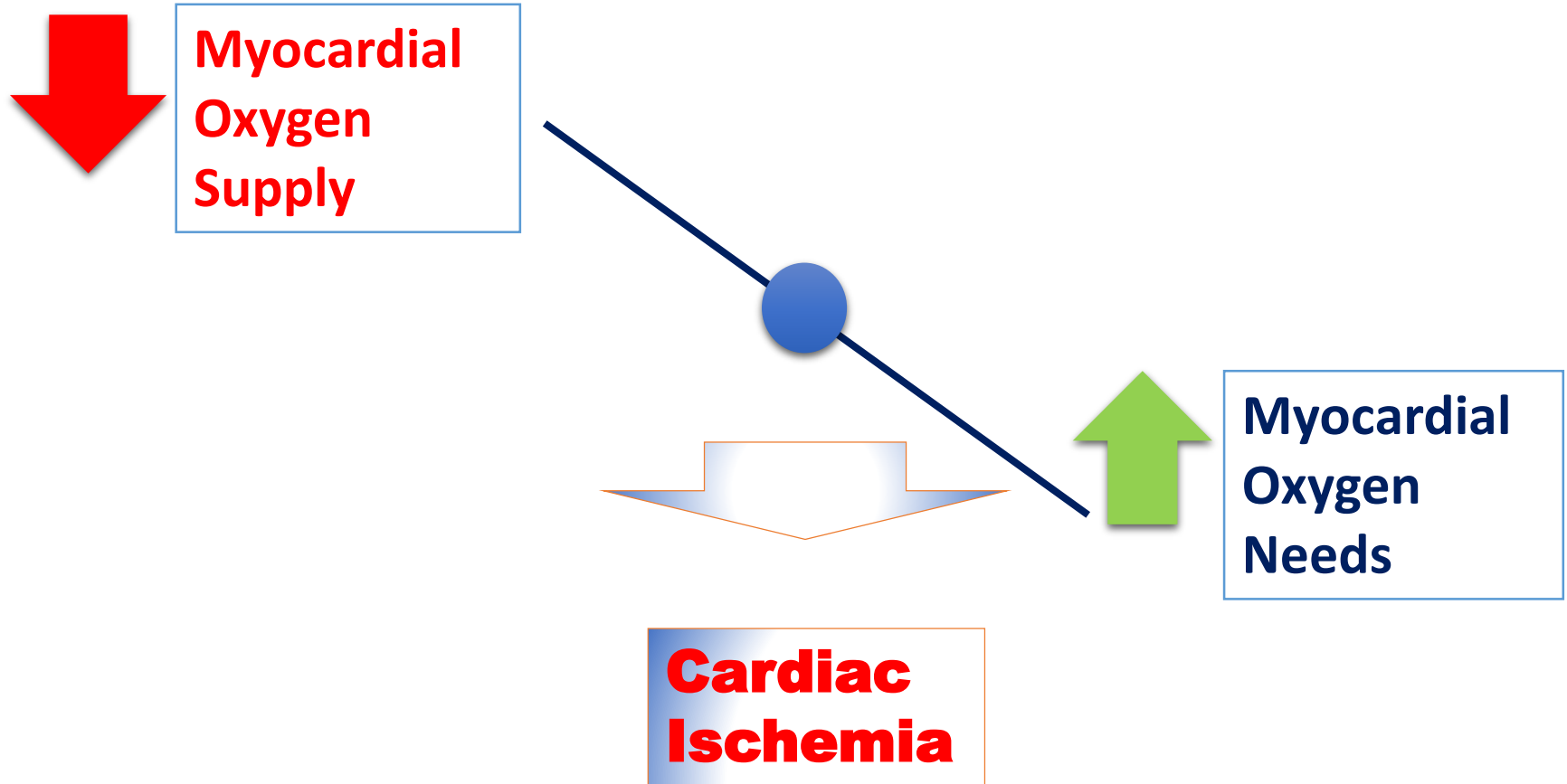


Aetiology

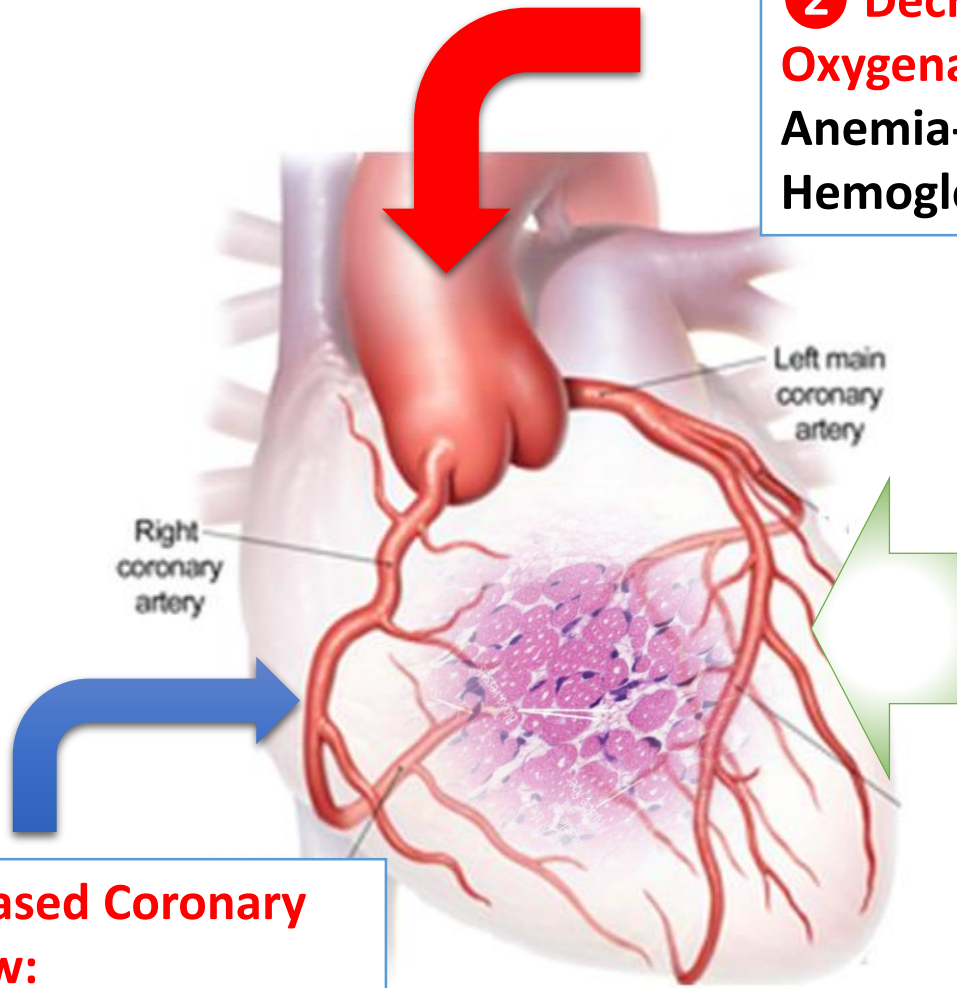
The condition is most likely caused by Coronary Heart Disease (i.e. **Atherosclerosis of the coronary arteries**)



Predisposing Factors (1)



Predisposing Factors (2)



② Decreased Myocardial Oxygenation:

Anemia- LCO- Lung Diseases- Hemoglobin Defect

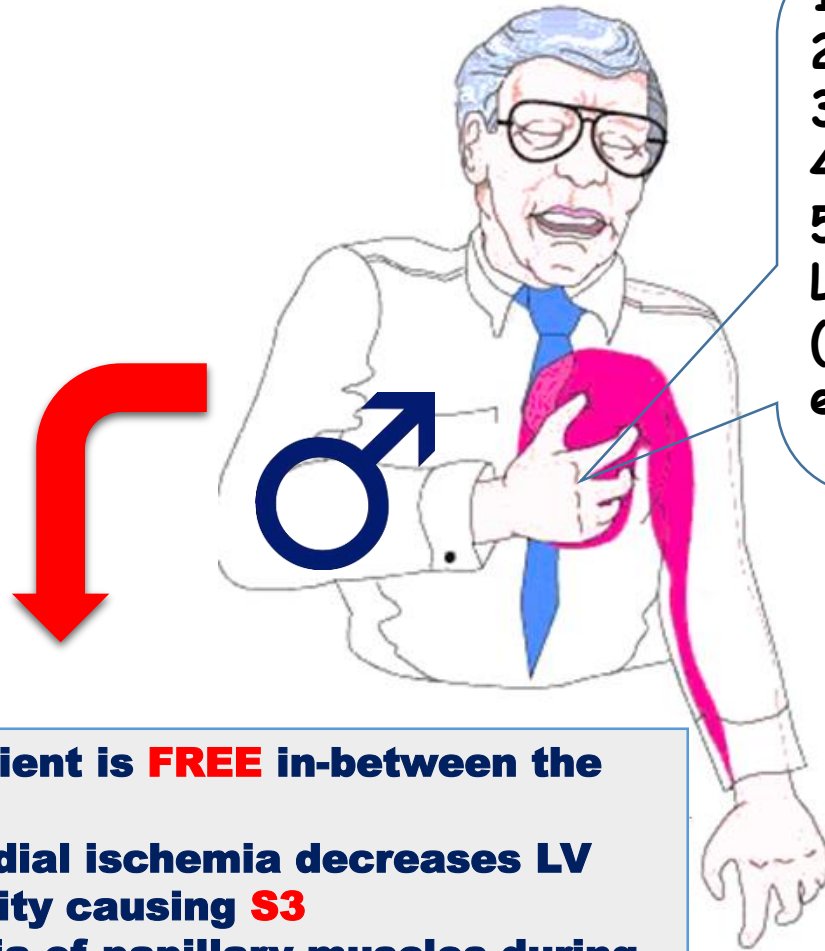
③ Increased Myocardial Oxygen Needs:

Thyrotoxicosis- Aortic Stenosis-  BP, and increased heart rate e.g. exercise or sexual intercourse

① Decreased Coronary Blood Flow:

Atherosclerosis- Coronary Arterial Stenosis- Coronary artery Spasm

Clinical Picture

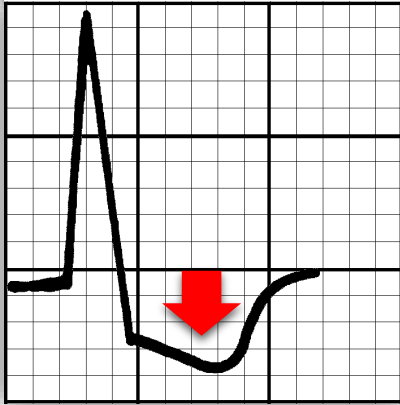


- 1- Retrosternal
 - 2- Compressing
 - 3- Radiating to left shoulder
 - 4- ppt by exercise or exertion
 - 5- Relived by rest or nitroglycerin
- Lasts few minutes to 15 minutes
(?! The attack may end by eructation # GIT problems)

- ① The patient is **FREE** in-between the attacks
- ② Myocardial ischemia decreases LV distensibility causing **S3**
- ③ Ischemia of papillary muscles during the attack may cause **Murmur like MR**
- ④ LV dysfunction during the attacks may cause **Paradoxical Splitting**
- ⑤ Reflex sympathetic ++++ due to the pain may lead to VC thus causing **pallor of skin + ⬆ BP**

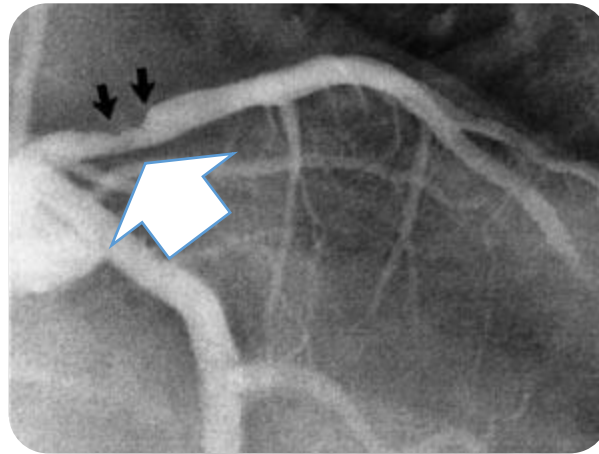
Special Investigations

ECG

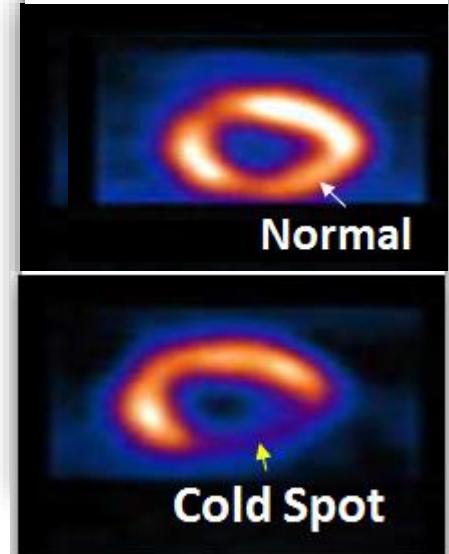


Depressed ST Segment

Coronary Angiography



Nuclear Scanning

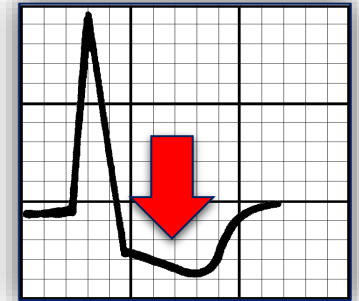
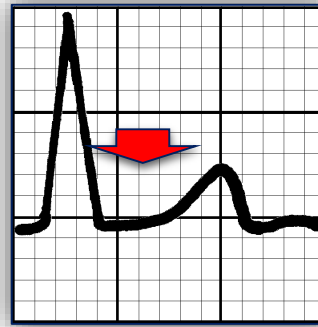
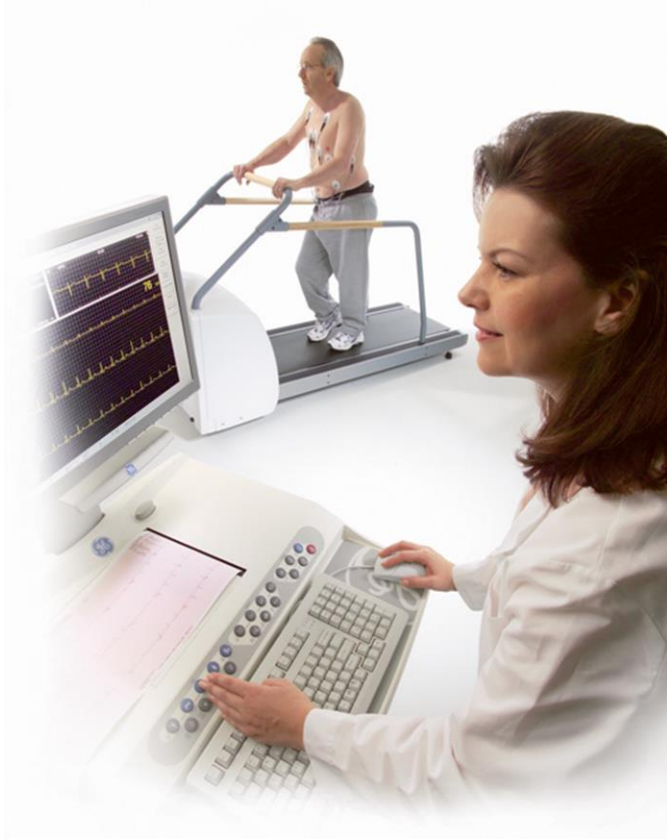


Stress Echocardiography may detect segmental wall motion abnormalities (During Exercise)

To detect the cause



- 1- ECG.....LVH
- 2- Thyroxin Level
- 3- CBC>>>>>Anemia
- 4- Serum Lipids..... LDL Cholesterol



Depression of the ST segment of more than 2 small squares is significant.



Treatment

- ① treatment of any aggravating factor
- ② treatment of the acute attack by Sublingual Nitroglycerin
- ③ Prophylaxis against the attacks by giving Nitroglycerin 5 minutes before any activity that may provoke an attack
- ④ Prevention of the attack by Long acting Nitroglycerin- Beta Blockers- Ca Channel Antagonists
- ⑤ Antiplatelet Drugs e.g. Low dose Aspirin (325 mg/day) to decrease *atheromatous* thrombus formation.

What is Prinzmetal's Angina Variant?

- 1 Due to Coronary artery spasm
- 2 More common in females
- 3 Precipitated by smoking
- 4 ST Segment is Elevated
(But **No increase in CPK-MB**)

Never use beta blockers
as this may precipitate an
attack.

Treatment is by
Nifedipine and
Nitroglycerin

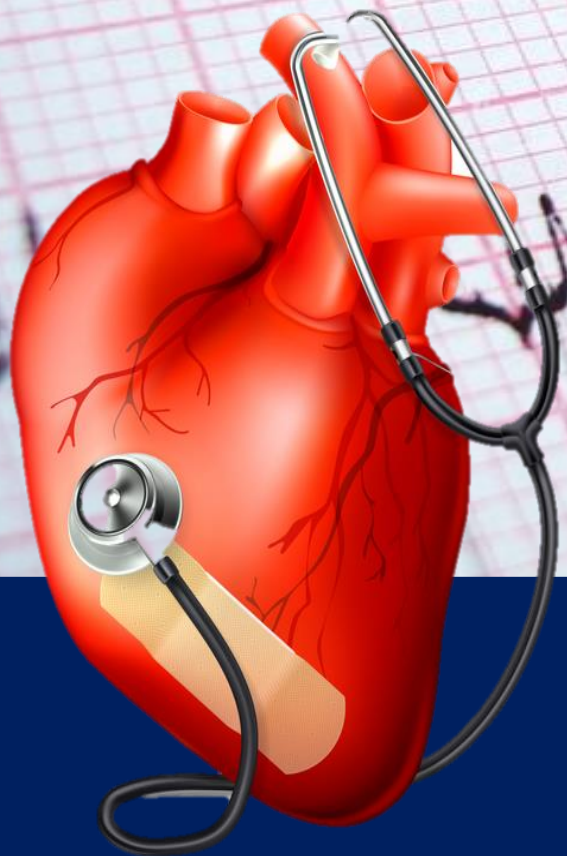
During
the attack



After
treatment



Cardiology



Acute Myocardial Infarction

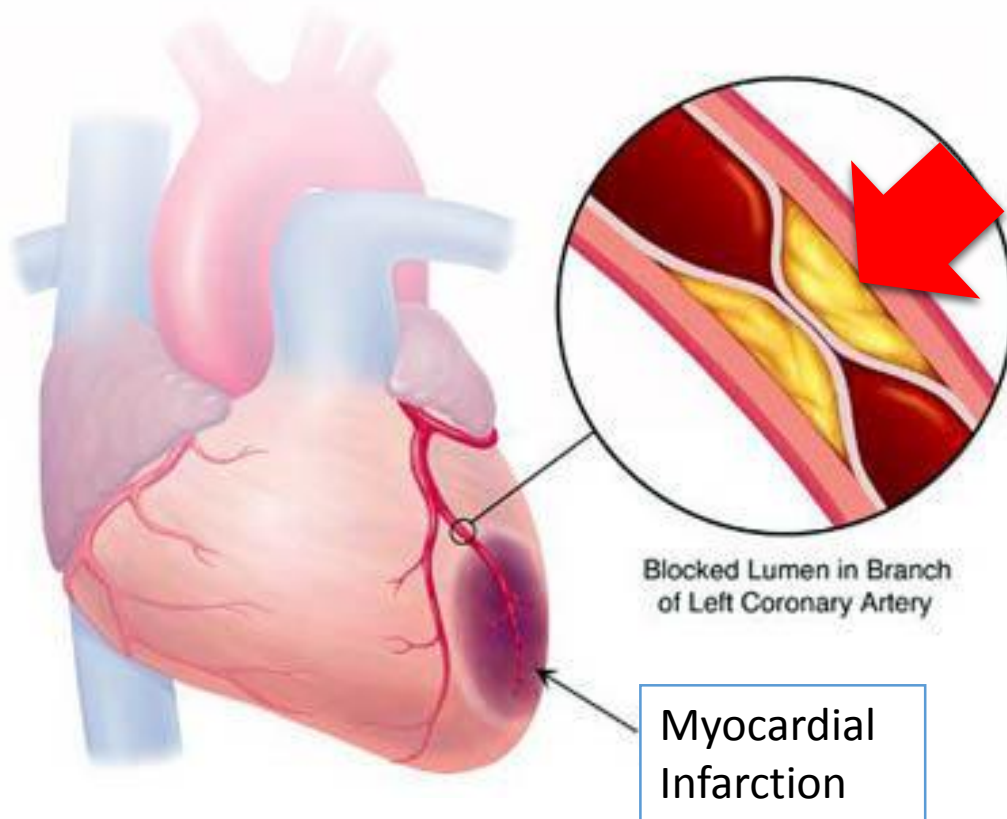
Definition

Death of part of the Myocardium due to +++++ Myocardial ischemia.

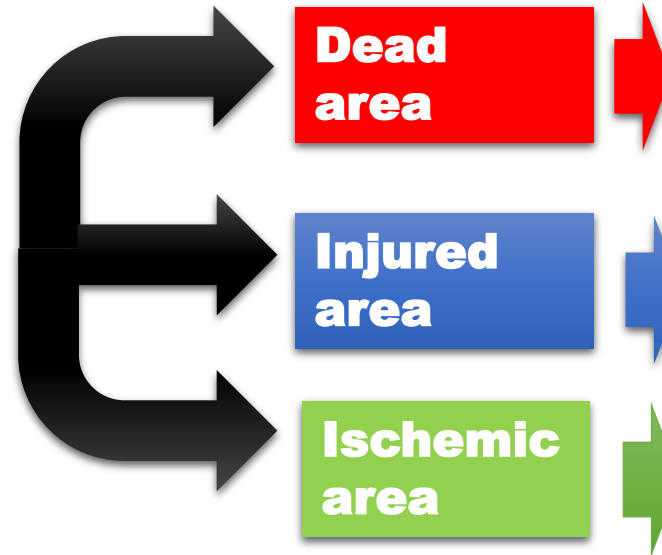
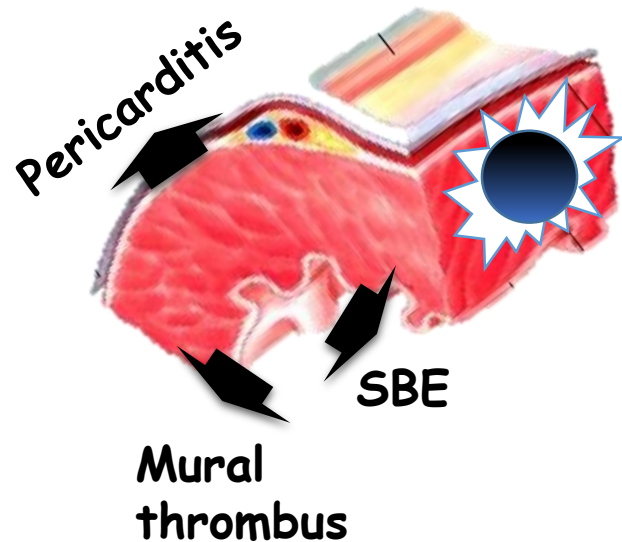


Aetiology

The condition is most commonly caused by an atheromatous thrombus causing **complete occlusion of the coronary arteries**.



Clinical Picture

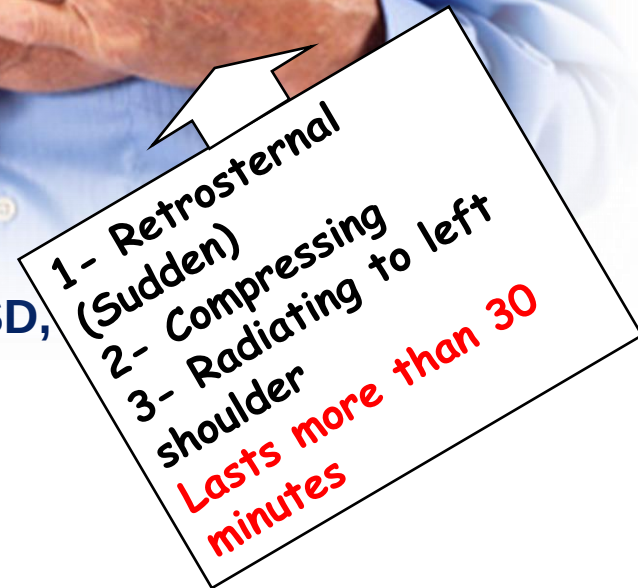


- ① Release of mediators: Pain
 - ② LV Dysfunction
 - ③ Necrosis of the dead myocardium (AMR- VSD- LBBB)
Arrhythmias
- Post Myocardial Infarction Angina

Clinical Picture

Death of part of Myocardium can cause:

- ① Release of mediators.....**Pain**
(Similar to angina pain *but* **More than 30 minutes**-
NOT related to exertion – occurs suddenly)
- ② LV Dysfunction.....
HF + Cardiogenic Shock
- ③ Necrosis of the dead myocardium can lead to
Catastrophic Complications
e.g. Acute MR (due to rupture of Papillary Muscles), VSD,
LBBB, Acute Cardiac tamponade
- ④ Myocardial electrical instability.....**Arrhythmias**
- ⑤ Irritation of the Pericardium**Pericarditis**
- ⑥ Mural thrombi can develop over the necrotic
endocardium....**Embolization**
- ⑦ **SBE** may develop over the dead endocardium



Special Investigations

ECG



- 1 Deep Q
- 2 Elevated ST Segment
- 3 Depressed T wave



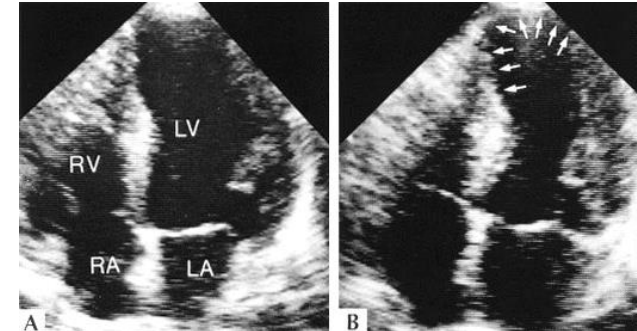
Serum Enzymes

CPK-MB

Other Enzymes:

Troponin T
Troponin I

IMAGING



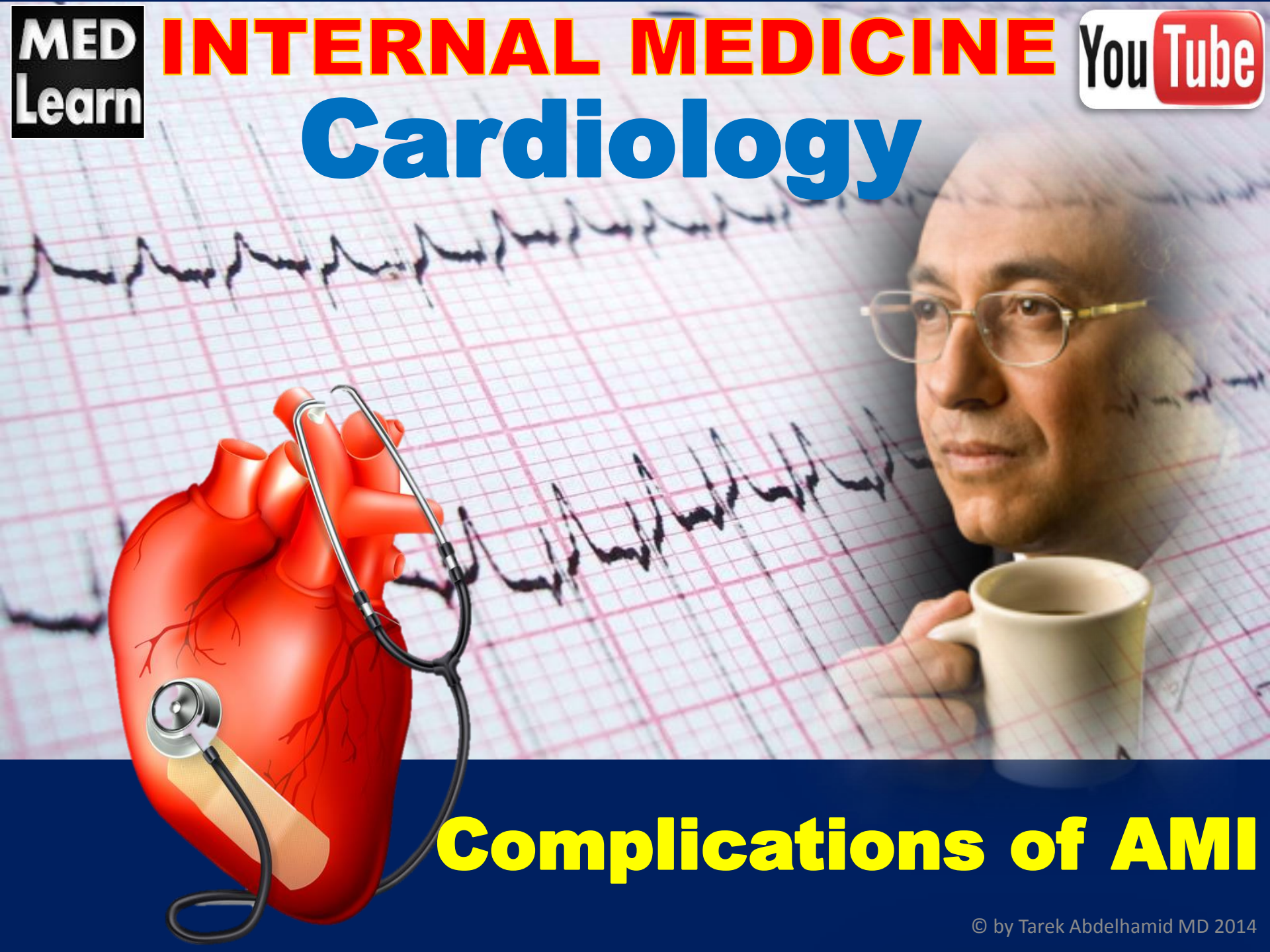
Segmental Wall Motion Abnormalities by Two-Dimensional Echocardiography

Treatment

- 
- ① Morphine SO₄ (to decrease Pain)
 - ② Aspirin (low dose) to decrease platelet aggregation
 - ③ Anticoagulants : Mini dose heparin
 - ④ Thrombolytic therapy by **t-PA** or Streptokinase **in the first 4-6 hours of the Pain**
- Note: Streptokinase causes generalized effect which may lead to bleeding
- ⑤ Nitroglycerin to dilate the collateral circulation
 - ⑥ Oxygen should be given to correct any associated hypoxia
 - ⑦ Treatment of arrhythmias
 - ⑧ Recently GIK (Glucose-Insulin-K) was shown to have promising effect
 - ⑨ Beta Blockers
(to decrease myocardial O₂ needs)
 - ⑩ ACEI (to decrease the load on the heart)

Note: Stool softeners may be used to decrease straining during defecation which may decrease CBF.

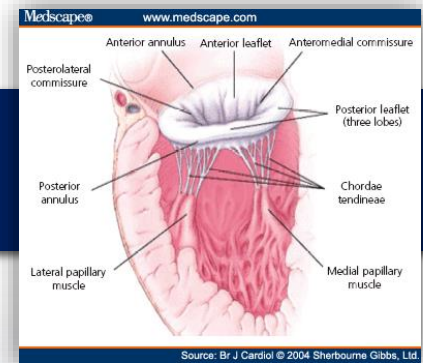
Cardiology



Complications of AMI

Condition (1)

Acute MR



Treatment:
Vasodilator therapy +
IABCP + Surgery

Comments:
1- Vasodilator therapy includes IV Nitroglycerin or Nitroprusside
2- Systolic Murmur of MR
3- May lead to APO
4- *Two Dimensional ECHO* with Doppler imaging is the investigation of choice



Condition (2)

Cardiogenic Shock



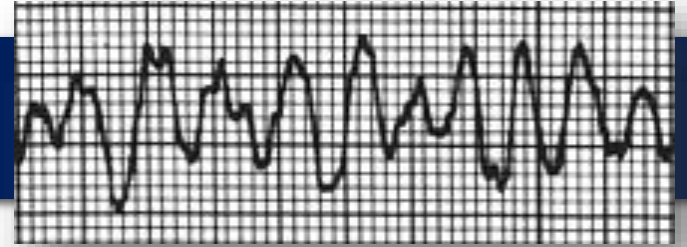
Treatment:
Dopamine

Comments:
All patients should be evaluated for surgically correctable cause e.g. AMR or VSD



Condition (3)

VT



Treatment:
Lidocaine

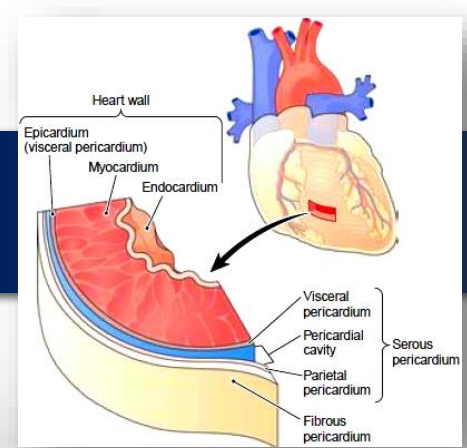
Comments:
One of the advantages of Lidocaine is that it does not inhibit Myocardial Contractility.



Condition (4)

Acute Pericarditis

Treatment:
Aspirin



Comments:
1- NSAID drugs and Corticosteroids are Contra Indicated as they inhibit myocardial scar formation thus predisposes to myocardial rupture.
2- NEVER give Heparin if you suspected pericarditis as it can cause bleeding inside the inflamed pericardium which may lead to Cardiac tamponade.

Condition (5)

Sinus Bradycardia



Treatment:

Atropine [if symptomatic]

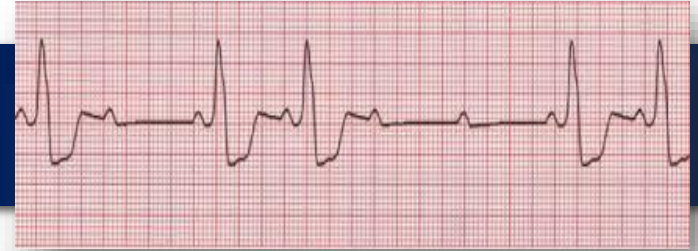
Comments:

Sinus Bradycardia is more common with **IW-AMI** (due to irritation of the nerve endings at the inferior surface of the heart → +++++ Vagal Stimulation thus causing ↓ HR)



Condition (6)

Heart Blocks



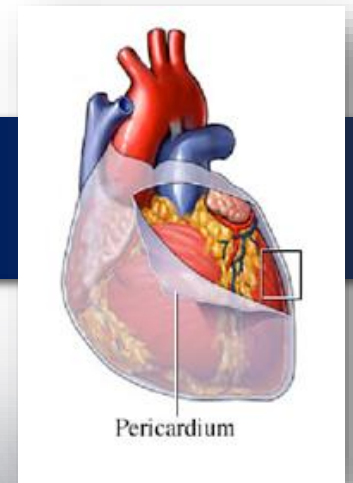
Treatment:
Pacemaker for life
threatening heart blocks

Comments:
e.g. CHB or Mobitz type II AV Block
(Occasional Dropped Beats)



Condition (7)

Dressler's Syndrome



Treatment:

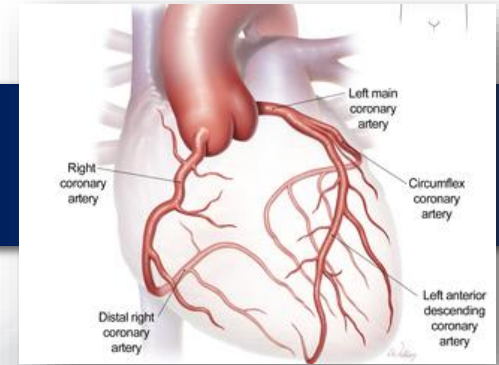
NSAI + Glucocorticoids

Comments:

- 1- Autoimmune reaction to cardiac proteins
- 2- May be due to release of cardiac proteins from the infarcted cells
- 3- **The antibodies may cause:** Pericarditis-
Pleuritis fever- Leukocytosis-  ESR
- 4- Typically occurs few weeks after the infarction
- 5-  **Anti-Myocardial Antibodies**

Condition (8)

Post-MI Angina

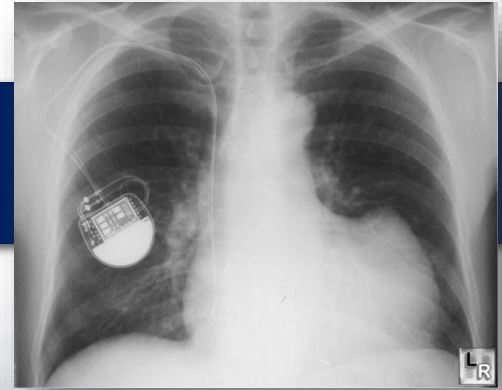


Treatment:
Aspirin + Nitrates + Beta
Blockers

Comments:
1- Correct underlying and contributing
factors e.g. D.M.- BP -
Hyperlipidemia
2- Coronary Angiography
3- For symptomatic PMI-SVT..... give
Amiodarone
4- For Post MI- Ventricular
arrhythmias....use Automatic
Implantable Cardioverter Defibrillator
[AICD]

Condition (9)

Cardiac Aneurysm



Treatment:
Mainly Surgical

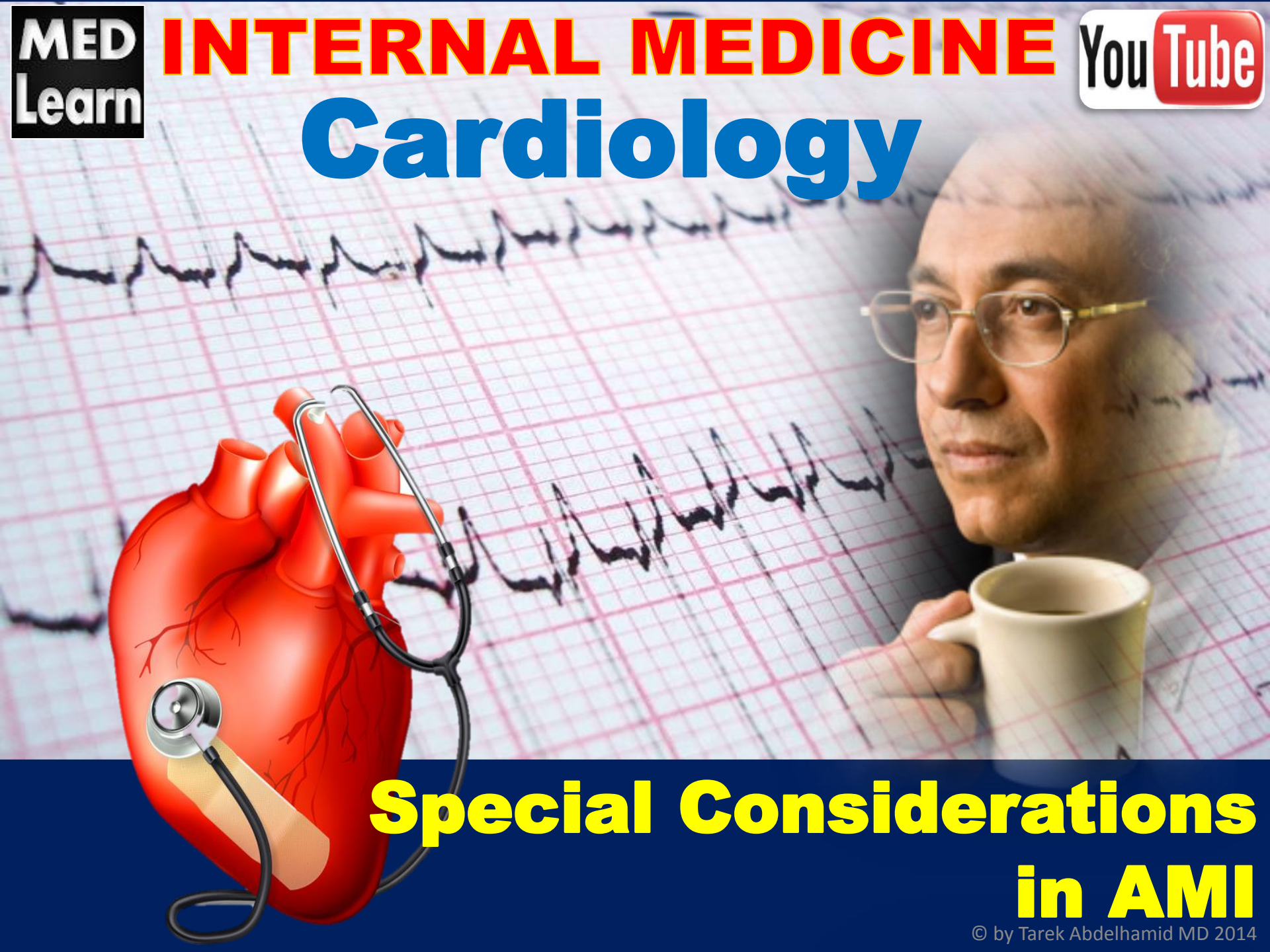


Comments:

- 1- Suspected if **Persistent ST segment Elevation** continued for 4-8 weeks after the infarction
- 2- May → Mural thrombi → arterial embolism (Needs **Anticoagulation**)
- 3- Echocardiography is Diagnostic

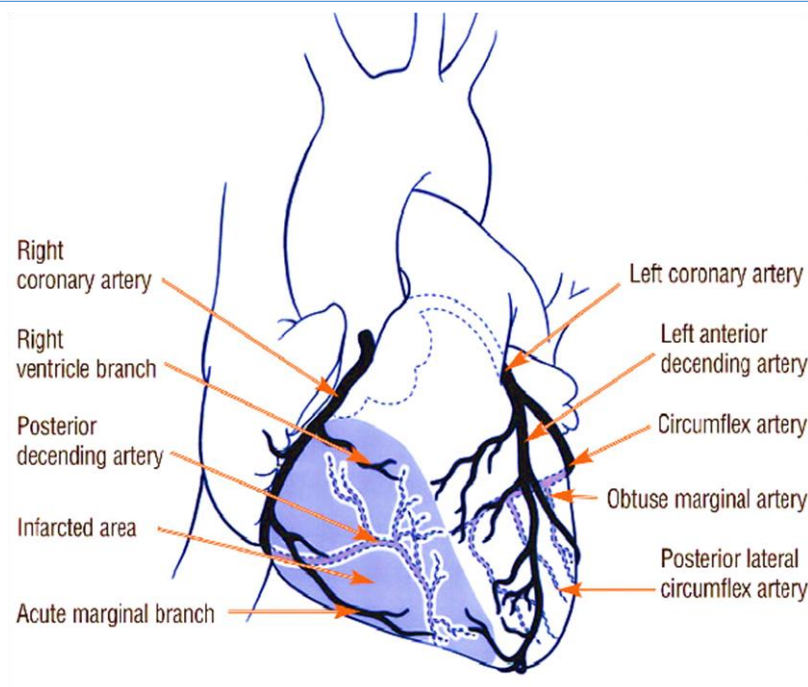
Suspect it when
ECG shows **Persistent ST
Segment elevation** for more
then 6 weeks after AMI

Cardiology



Special Considerations in AMI

Right Ventricular Infarction



① Associated with IW-AMI



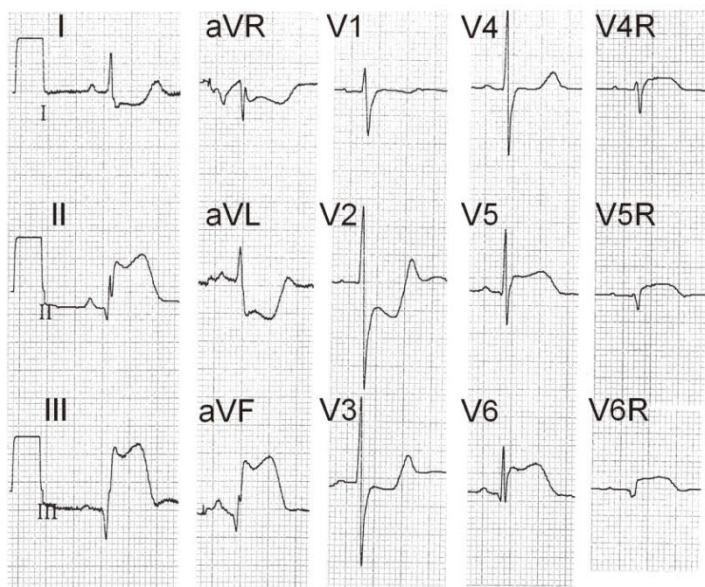
② Presents as Hypotension and raised JVP



③ ECG may show ST segment elevation in **V3R + V4R**

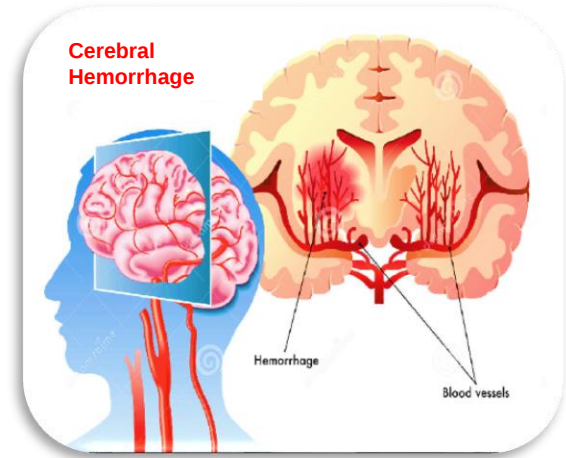
④ The hypotension may be aggravated by Diuretics- Nitrates- or Narcotics.

⑤ Treatment includes elevation of the legs and IV Saline



Complications of Fibrinolytic Therapy

The most dreadful complication of fibrinolytic therapy is **Cerebral Hemorrhage**



It is contraindicated in:

- 1 Increased tendency for bleeding (e.g. **Bleeding Disorders**)
- 2 When bleeding can cause serious complications (e.g. **Recent Stroke**)
- 3 When bleeding will be difficult to stop e.g. **Acute Pericarditis**-**Acute Peptic Ulcer**- **Active IBD**- **Active cavitating lung lesions**

Hemorrhage



- 1- **FFP**
- 2- Cryoprecipitate
- 3- Platelet transfusion

Other Points about AMI

① If nausea and vomiting occurred with using Morphine

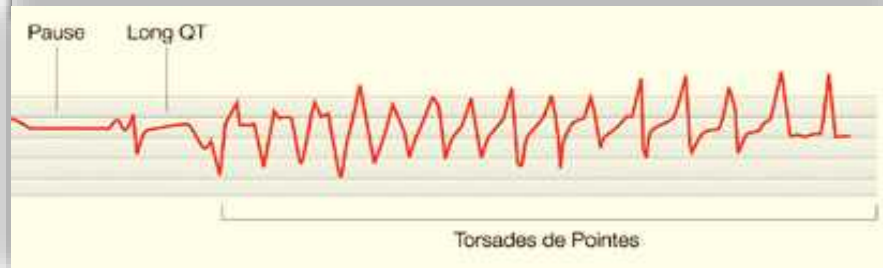


Antiemetic

② Agitated patients



IV Haloperidol (watch the QT interval as it may prolong it)

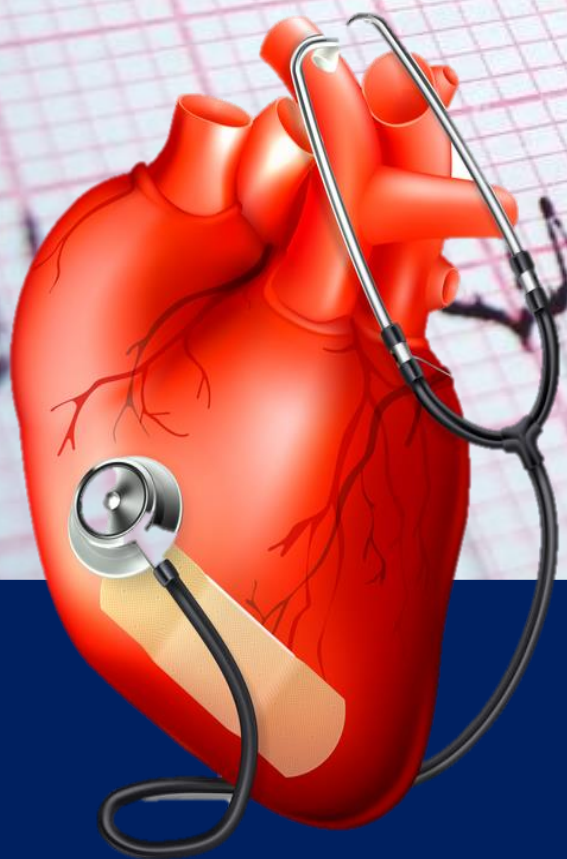


③ If Nitroglycerin caused reflex bradycardia



Atropine

Cardiology



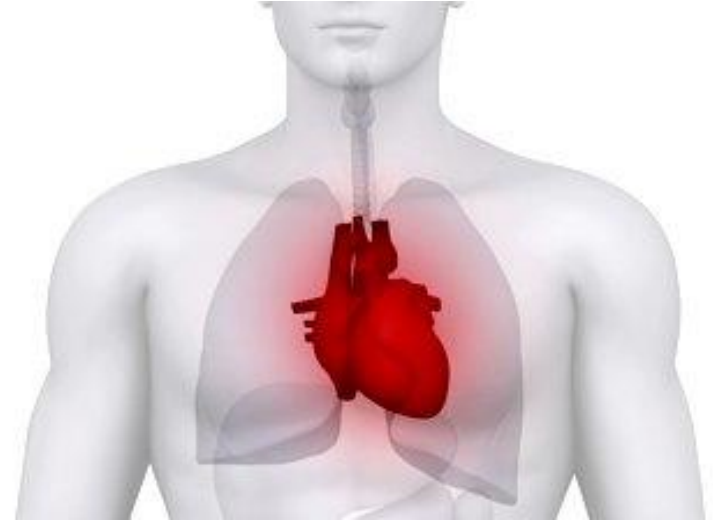
Heart Failure

Definition

Inability of the heart to pump blood sufficient for the metabolic needs of the body.

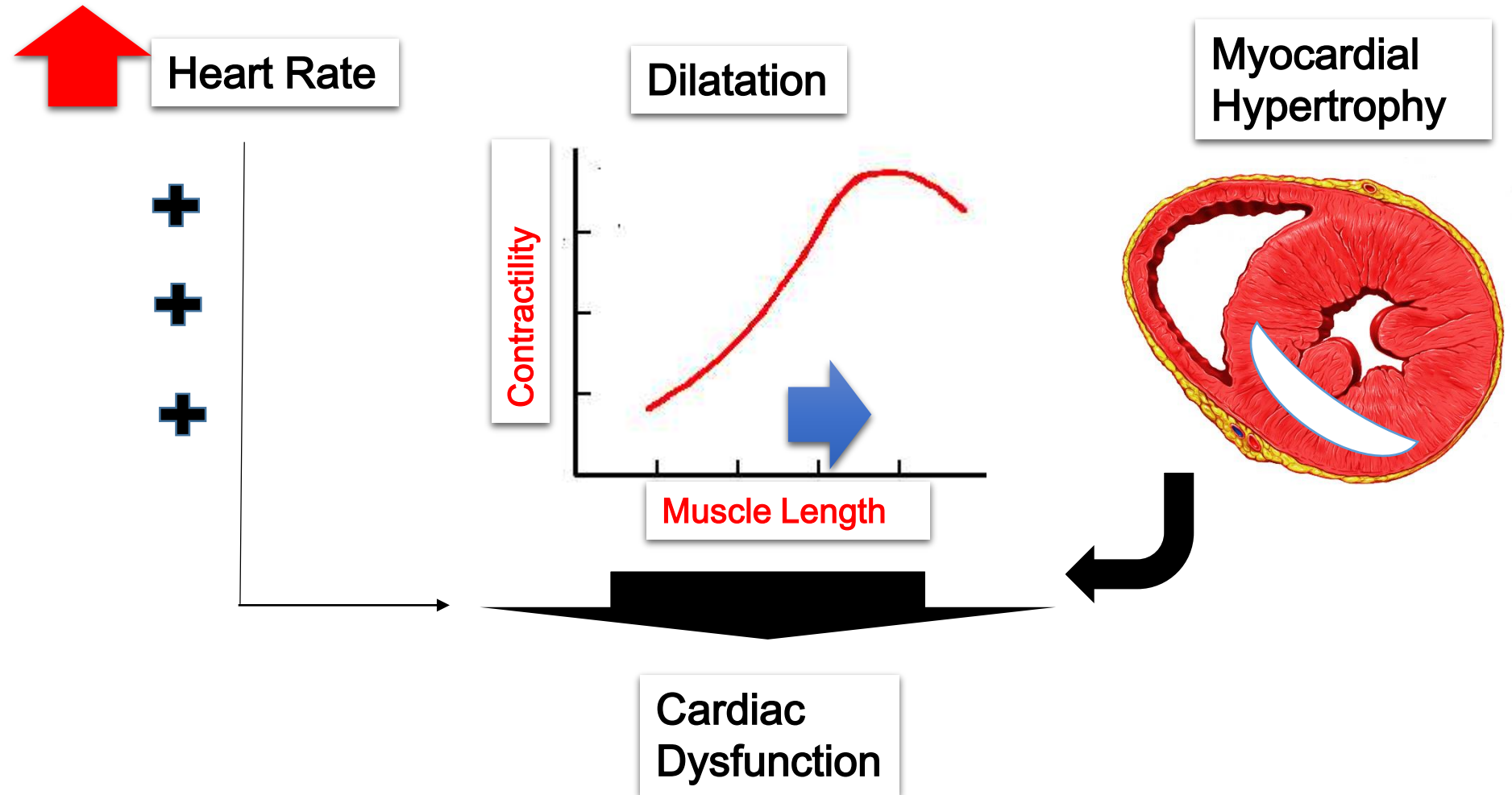
Aetiology

- ①  BP
- ② Ischemic Heart Disease
- ③ Cardiomyopathy
- ④ Valvular Heart Diseases
- ⑤ Congenital Heart Diseases

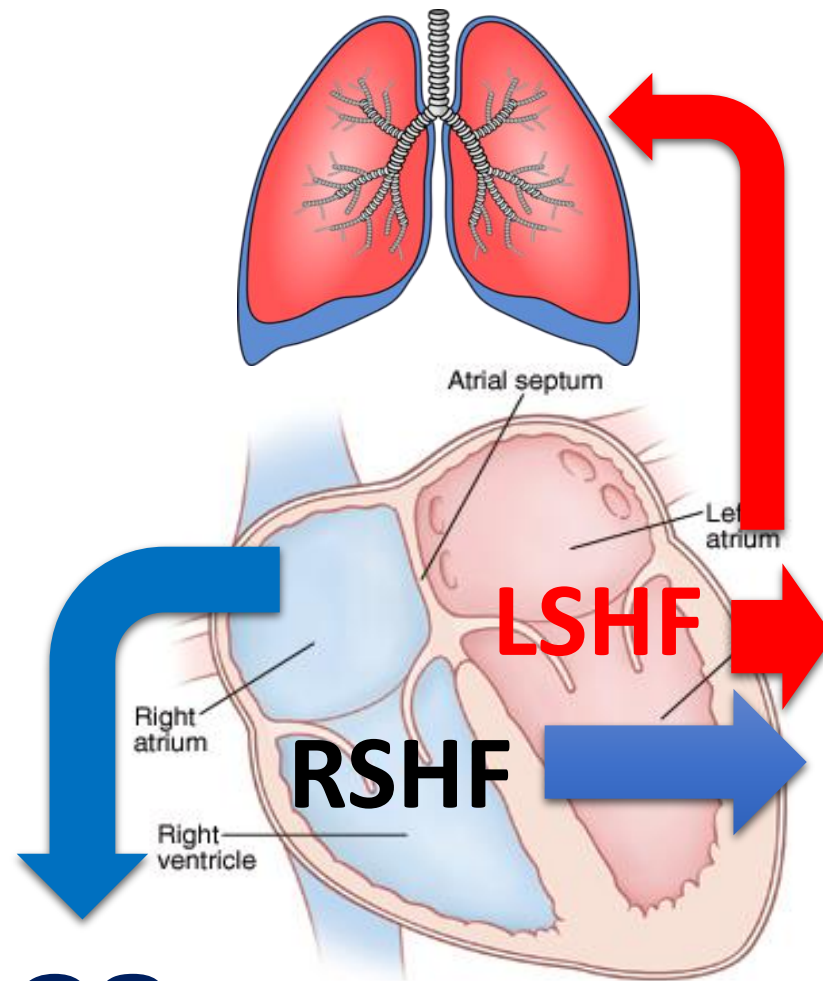


Note: Suspected cardiomyopathy when HF occurs without any obvious cause

Compensatory Mechanisms



Clinical Manifestations



LCS

Dyspnea- Orthopnea-
PND- APO- Cough-
Hemoptysis-
**Bilateral Basal Fine
Crackles**

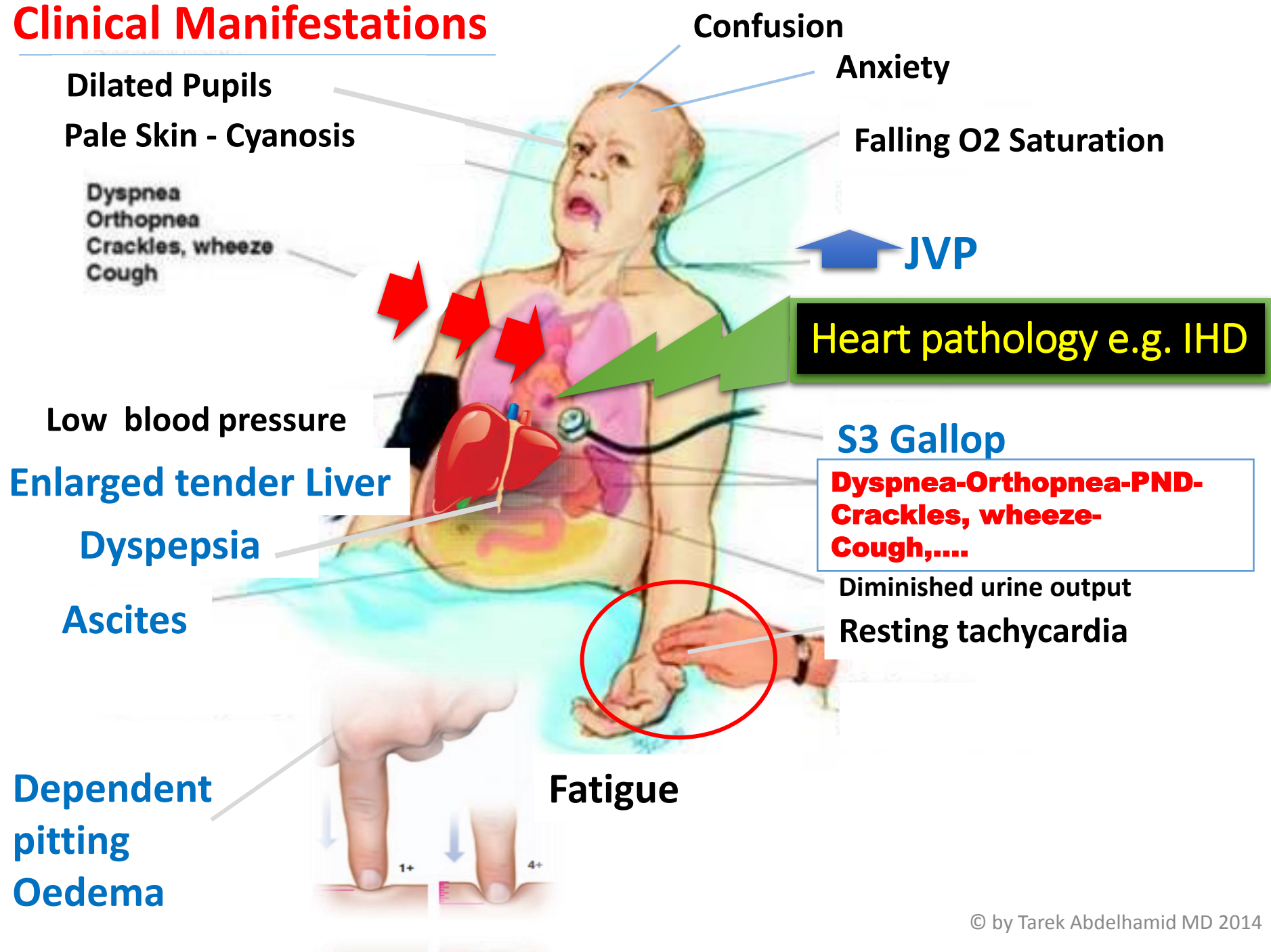
LCO

Headache- Dizziness-
Giddiness- Blurring of
vision- Syncopal attacks-
Oliguria - weakness
Confusion

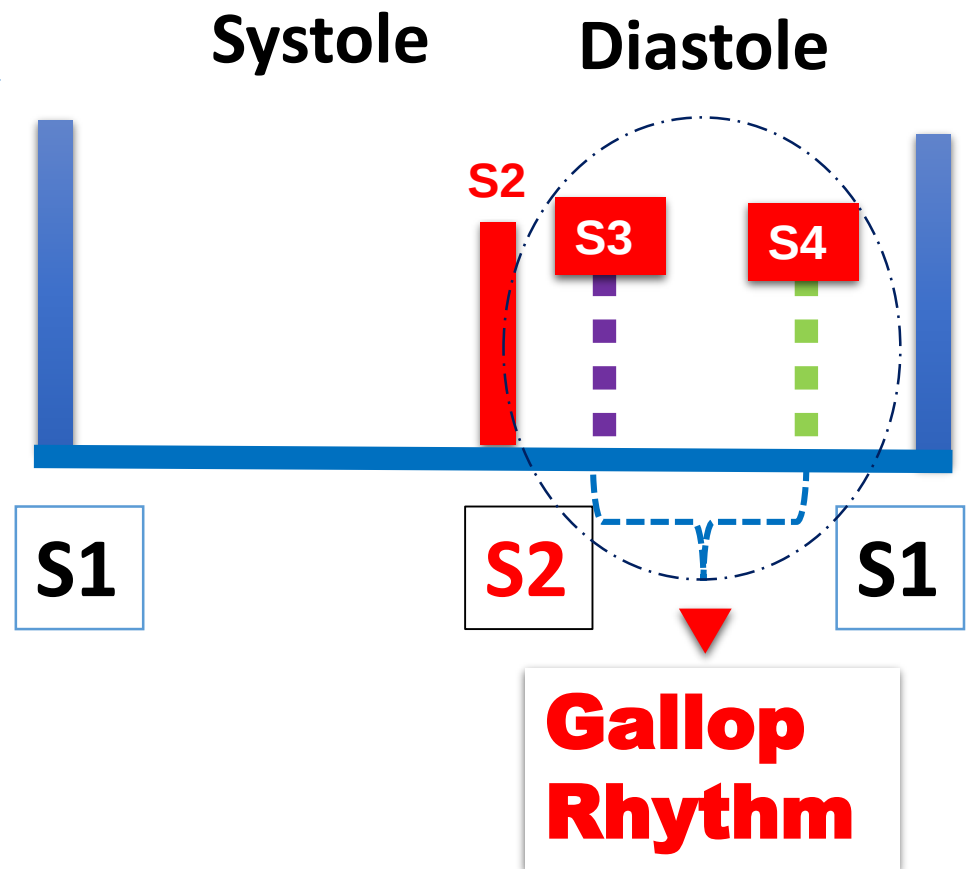
SCS

Oedema of both LL (less in
morning)- Dyspepsia to all types of
food- Enlarged tender Liver-
Congested Pulsating Neck Veins

Clinical Manifestations



Auscultation

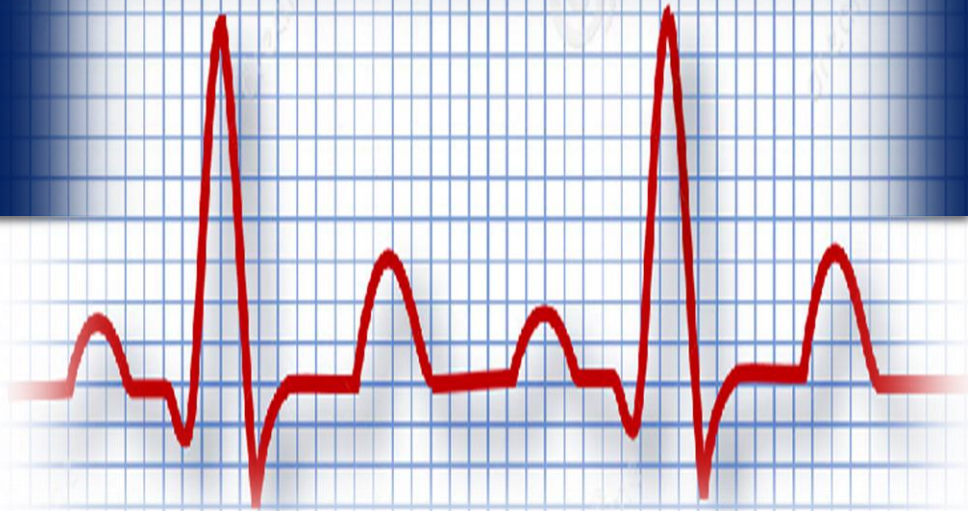


- 1- Functional Murmurs o MR and TR
- 2- Underlying Heart Problem
- 3- Gallop Rhythm

Note:

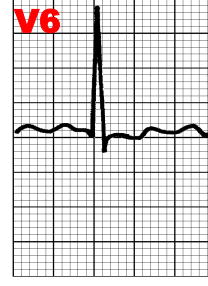
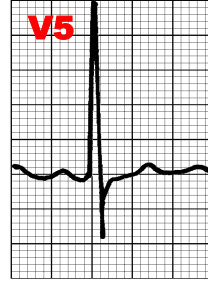
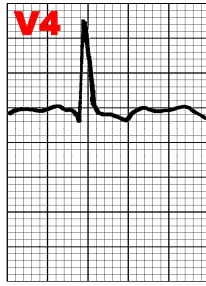
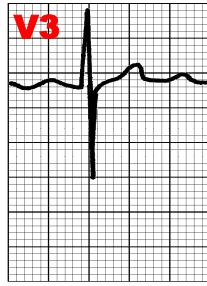
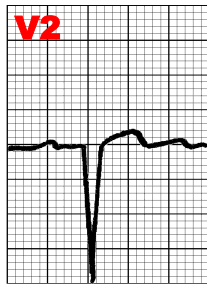
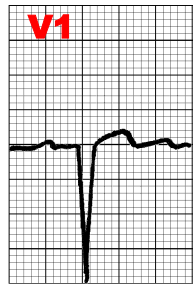
Heart failure with Bradycardia may be seen in the following conditions

- ① **Hypothyroidism**
- ② **CHB**
- ③ **Patients under Digitalis**



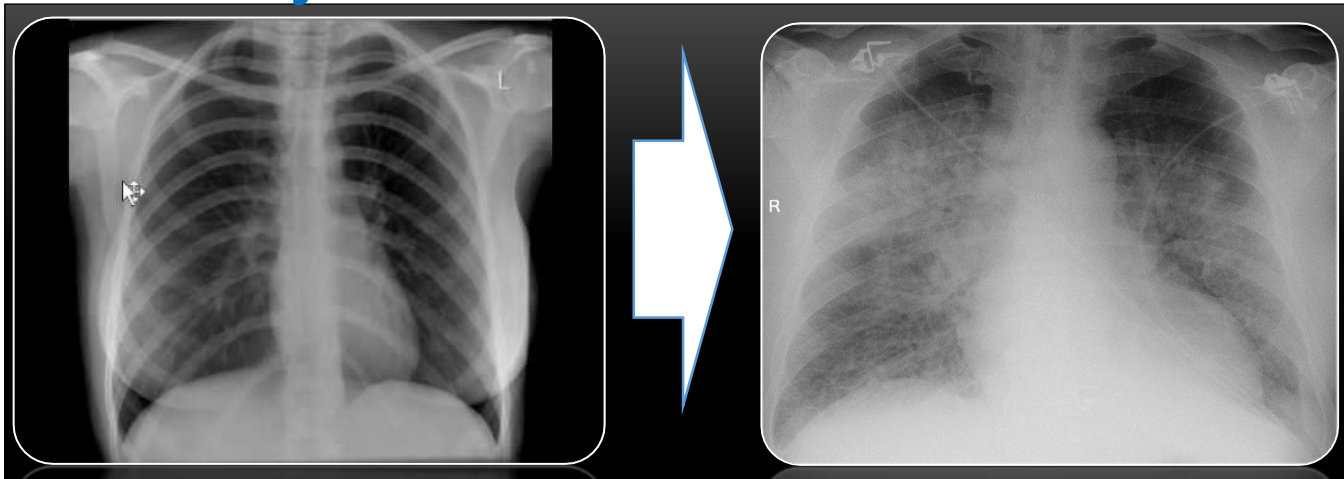
Special Investigations

① ECG:



LVH

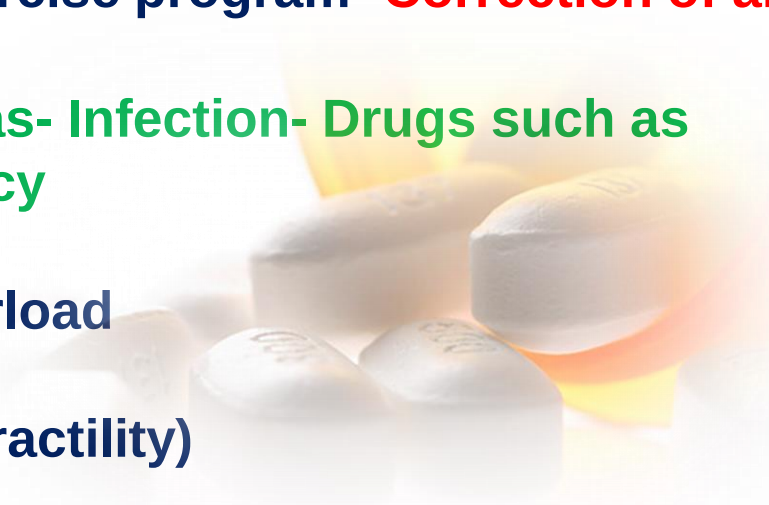
② X-Ray



③ Catheter: EDP

④ Echo: to determine cardiac size and functions

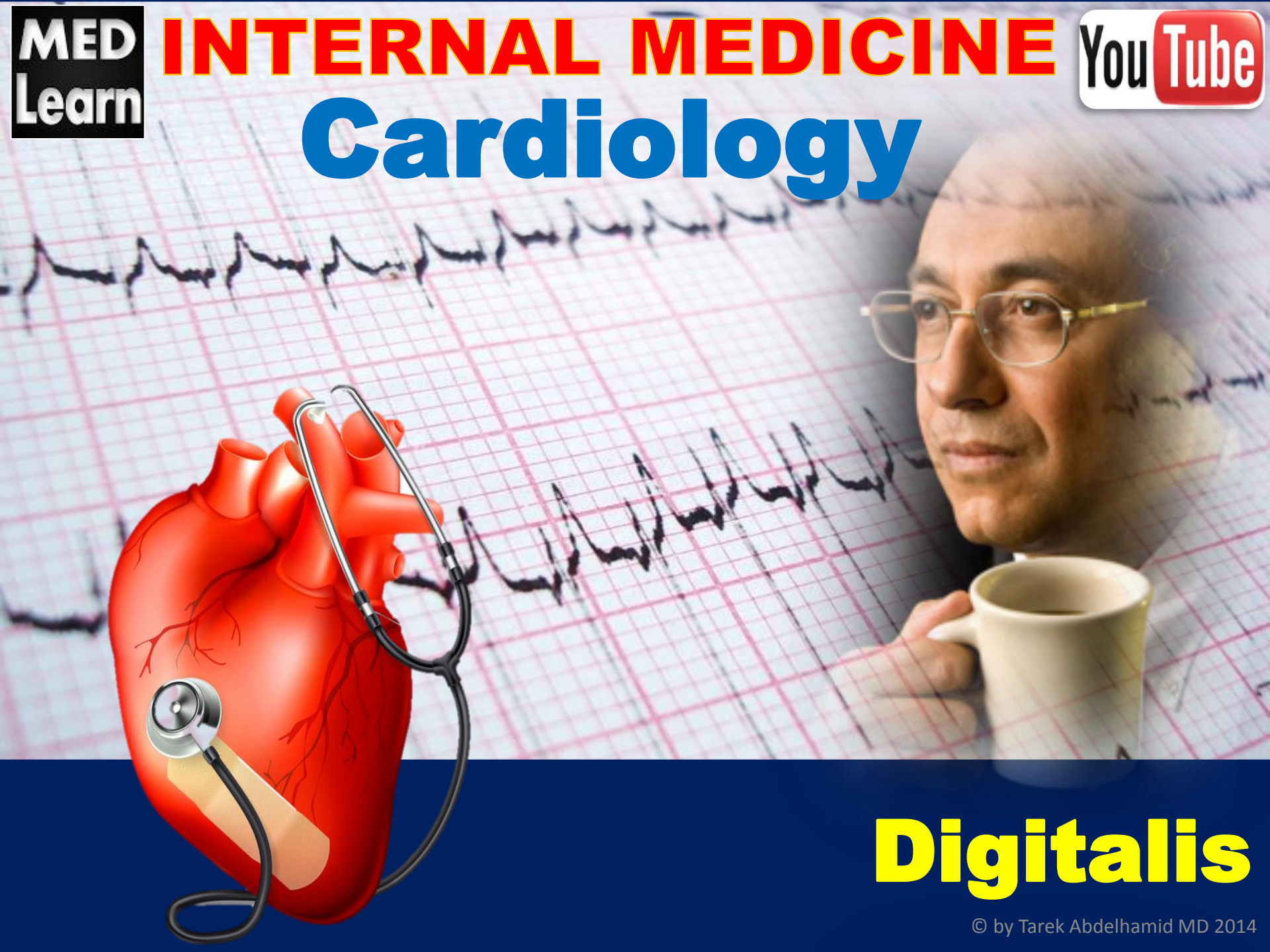
Treatment

- 
- ① Moderate salt restriction- Gradual exercise program- **Correction of any precipitating** or contributing factor
(e.g. Anemia- Thyrotoxicosis- Arrhythmias- Infection- Drugs such as NSAIs- high salt intake-Thiamine Deficiency)
 - ② Diuretics to decrease the volume overload
 - ③ Digitalis (to enhance myocardial contractility)
 - ④ Vasodilator therapy: e.g. Nitrates/ Hydralazine

Initial treatment usually include a combination of Diuretics plus ACE Inhibitors. If this combination was not sufficient to relief the symptoms you may add Digoxin.

- ⑤ Treatment of the cause
e.g. ttt of Hypertension- ttt of IHD-
Surgery for Valvular or congenital lesions

Cardiology

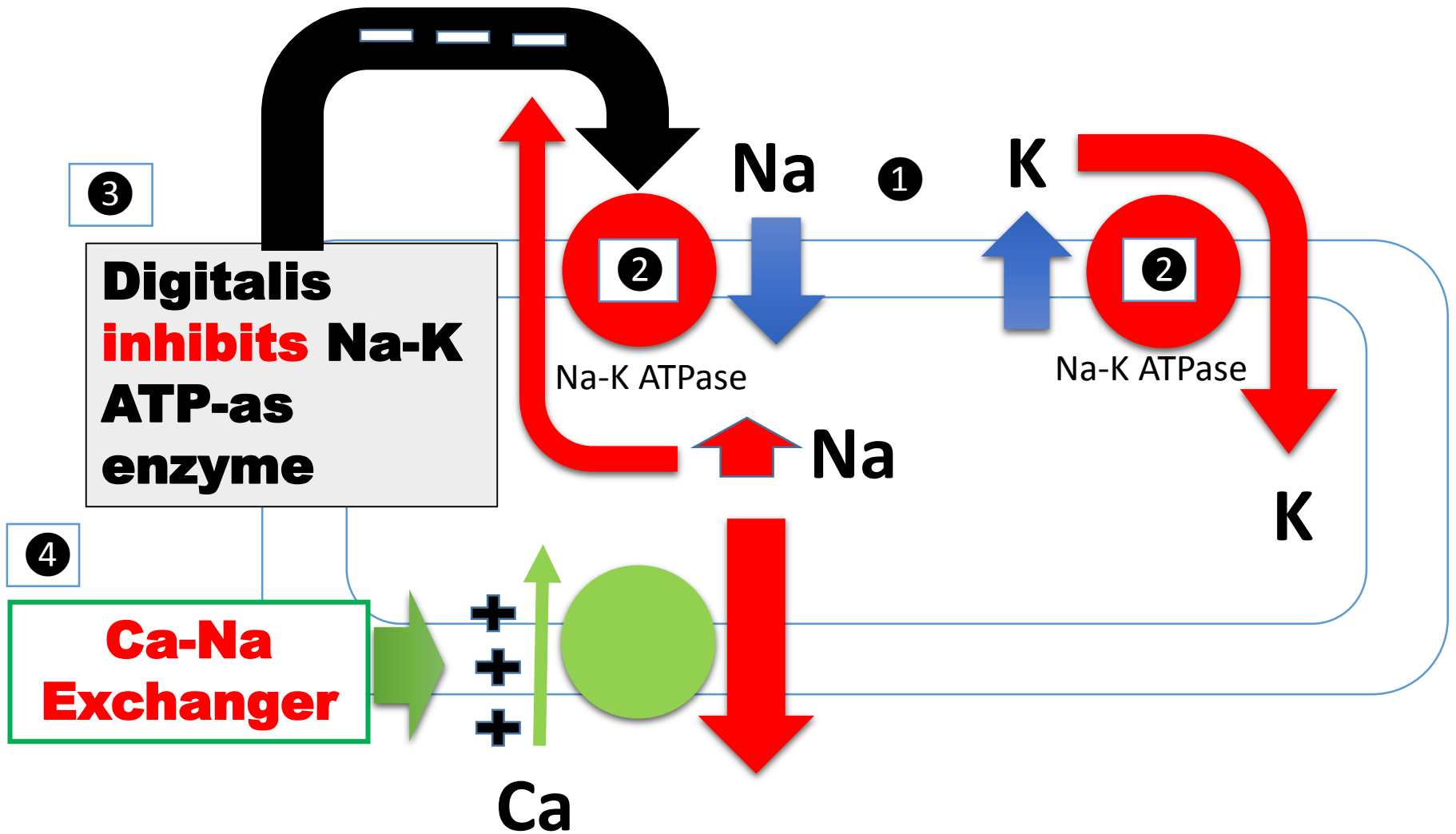


Digitalis

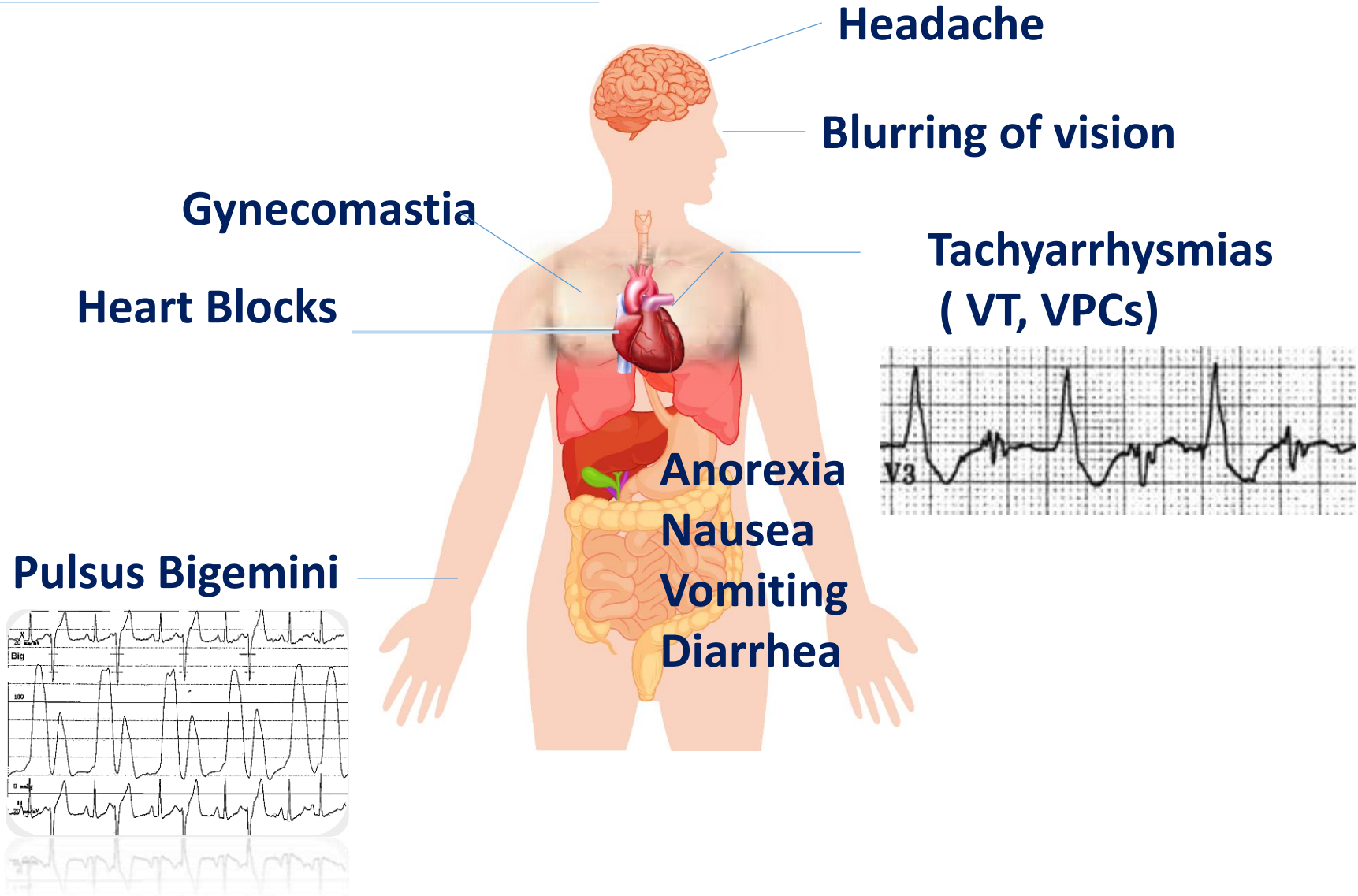
Mechanism



- ① Digitalis inhibits Na-K ATP-ase enzyme
- ② This inhibits Na pump thus causes accumulation of Na inside cardiac muscles
- ③ The accumulated Na is exchanged for Ca via Ca-Na Exchanger which results in accumulation of Ca inside the cardiac cells
- ④ The high Ca inside the cardiac increases Myocardial Contractility



Toxicity

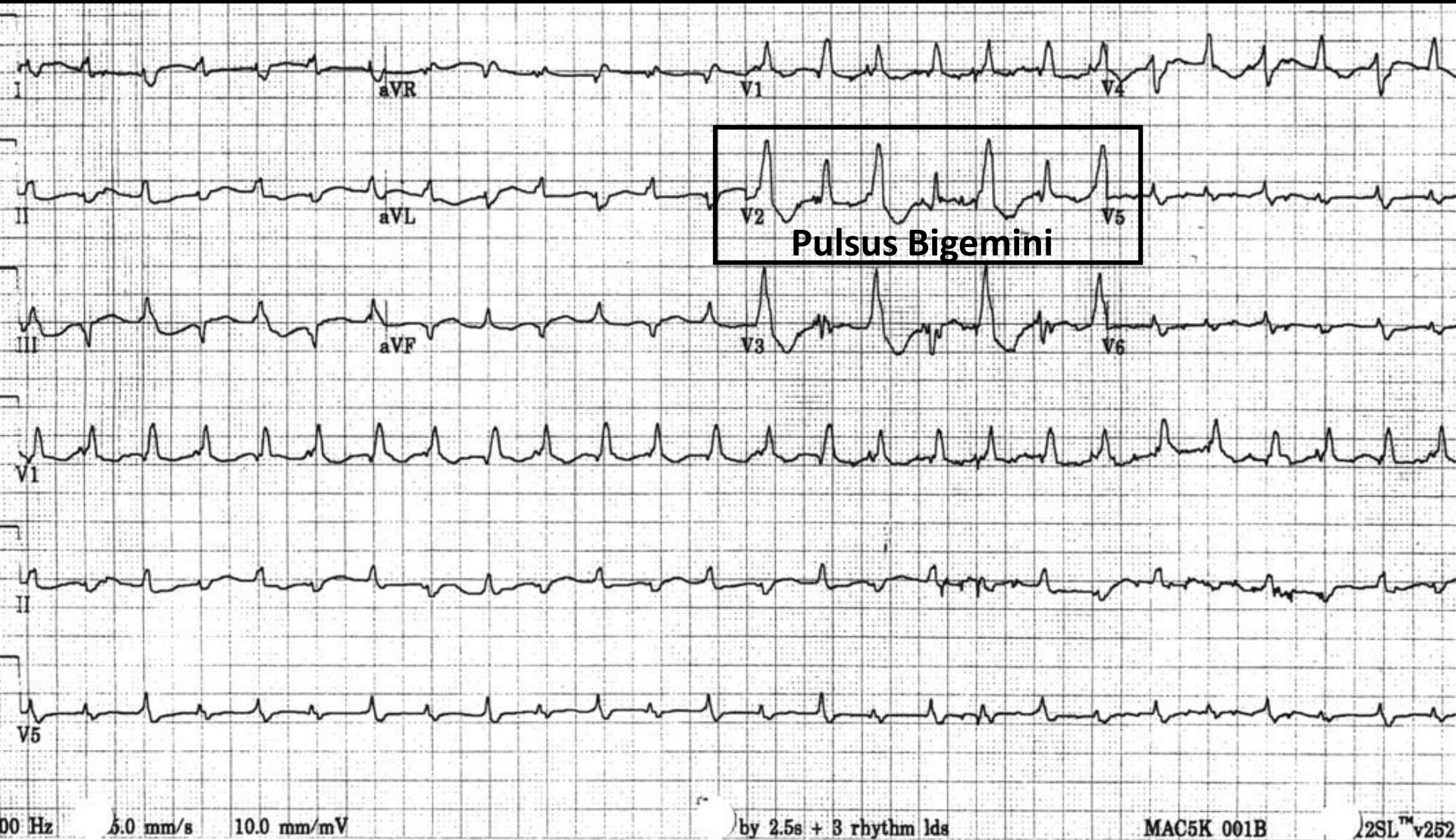


Predisposing Factors for Digitalis Toxicity



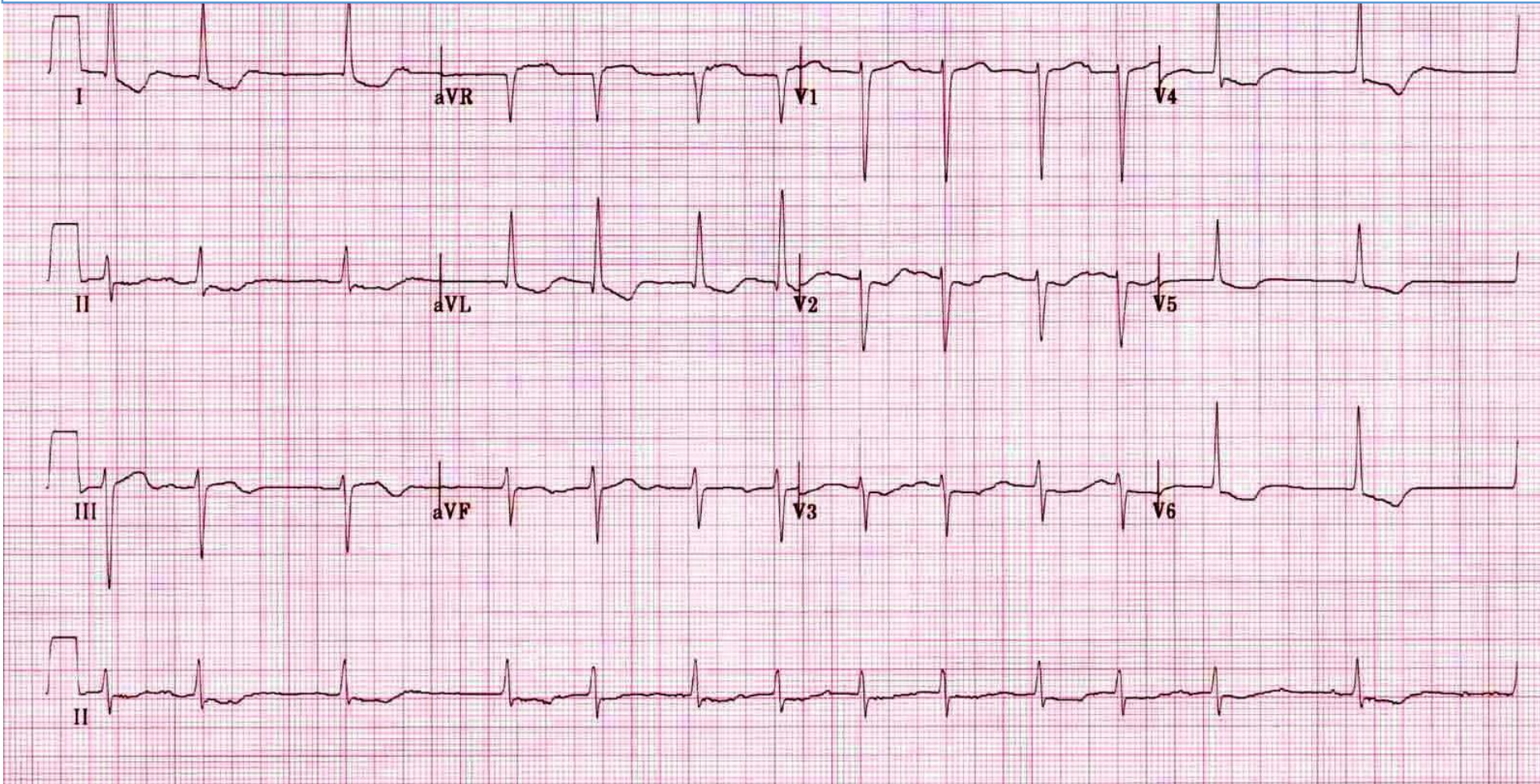
- ① Hypokalemia
- ② Hypercalcemia
- ③ Renal Failure
- ④ Volume Depletion (Hypovolemia)
- ⑤ Hypoxemia
- ⑥ Drugs: e.g. Amiodarone
- ⑦ Hypothyroidism

Digitalis Toxicity



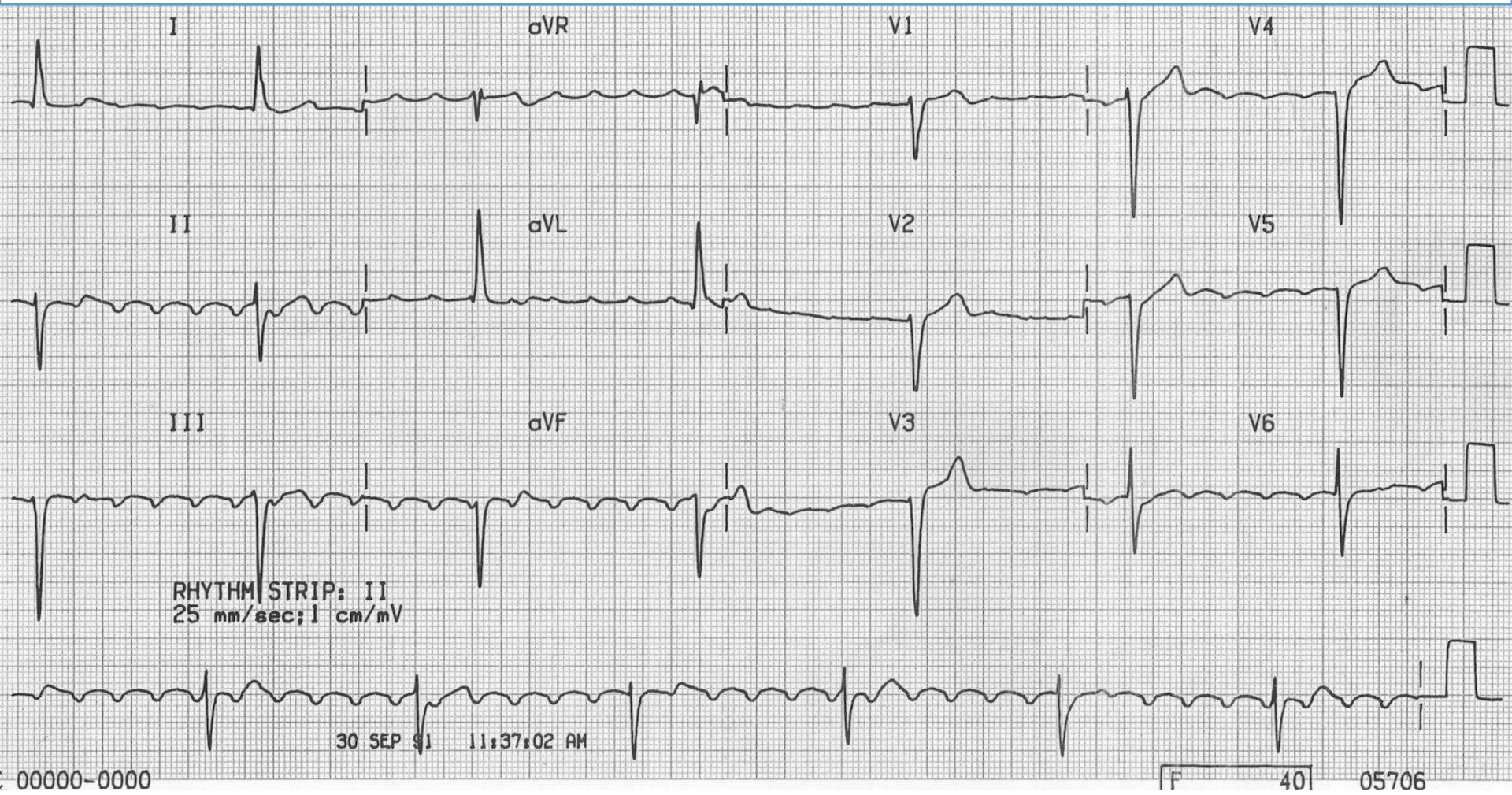
Digitalis Toxicity

Af plus digoxin effect scooping of ST



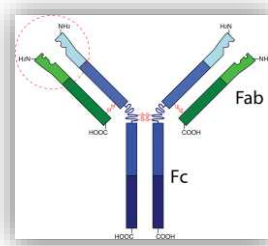
Digitalis Toxicity

Atrial flutter with AVB causing bradycardia



Treatment:

- ① **Stop Digitalis**
- ② **Correct any underlying P.F.: e.g. Hypokalemia**
(the latter could be caused by Diuretics)
- ③ If Ventricular Arrhythmias: Use Lidocaine or Phenytoin
(Avoid Quinidine- Amiodarone- Propafenone
as they increase Digoxin Level)
- ④ If Intractable Ventricular arrhythmia:
Use **Fab fragment of Digoxin Antibodies**
- ⑤ Treatment of Symptomatic Bradycardia is by
Atropine (as it may be due to increased vagal tone)



Treatment:

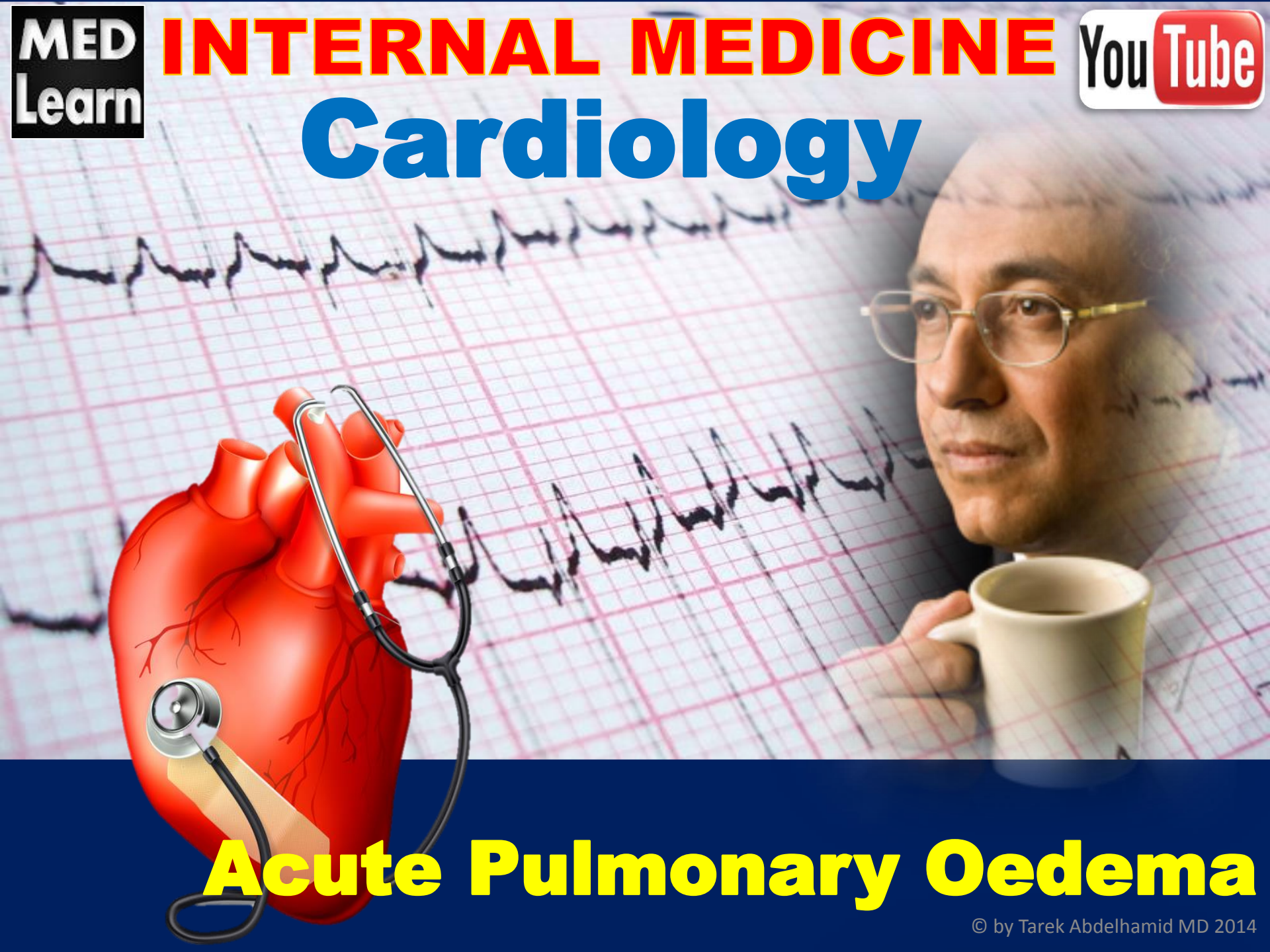
3 Important Notes

Note 1 Repeated doses of Fab fragment of Digoxin Antibodies are needed as its $\frac{1}{2}$ life is shorter than Digitalis

Note 2 Avoid DC shock as it may precipitate fatal VF

Note 3 Avoid Sympathomimetics for Bradycardias as they may worsen the ventricular arrhythmias.

Cardiology



Acute Pulmonary Oedema

تحذير هام

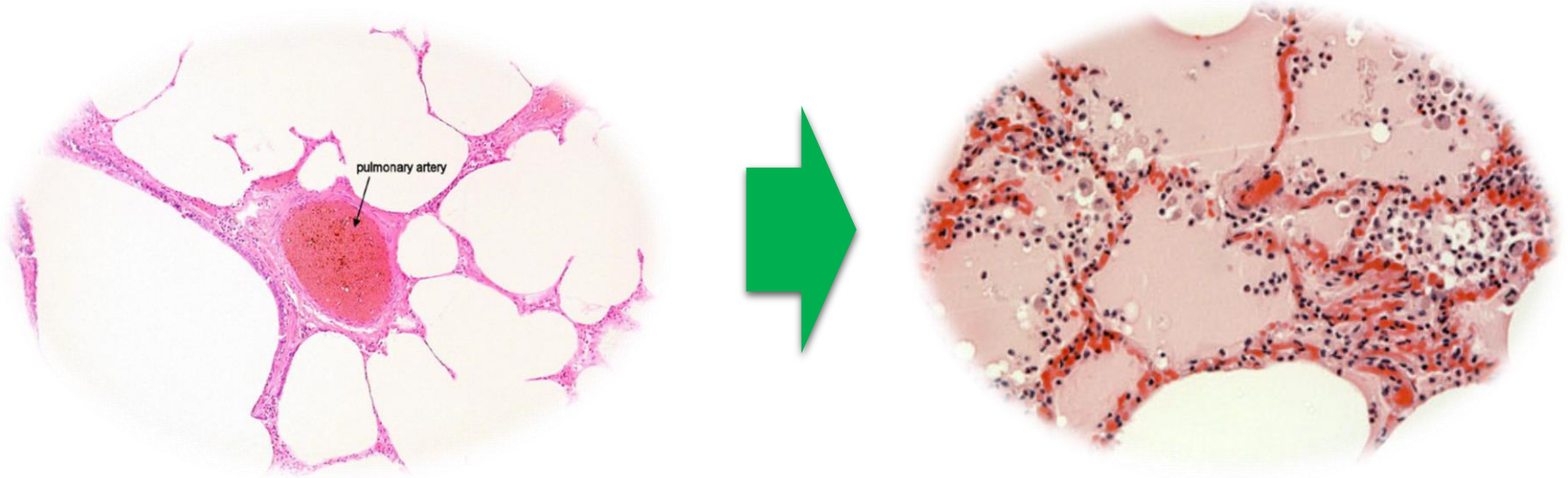
هذا الفيديو و جميع الرسوم التوضيحية المتاحة فى هذا الموقع هم تحت حماية قانون الملكية الفكرية

هذا الفيديو و جميع الرسوم التوضيحية المتاحة فى هذا الموقع هم تحت حماية قانون الملكية الفكرية و لا يحق لأحد إستخدامه أو إستنساخه أو إعادة طبعه أو طبع أجزاء منه بدون إذن مسبق من الدكتور طارق عبد الحميد (أو من ينوب عنه قانونياً). و من يتعدى على الملكية الفكرية المذكورة فسوف يتعرض للمسائلة القضائية عن جريمة إنتهاك حقوق الملكية الفكرية. و إستخدام الفيديوهات و المادة العلمية المذكورة مكفول فقط لمشتري واحد فقط و لأخذ تصريح إستخدام لأكثر من شخص برجاء الإتصال بالدكتور طارق عبد الحميد



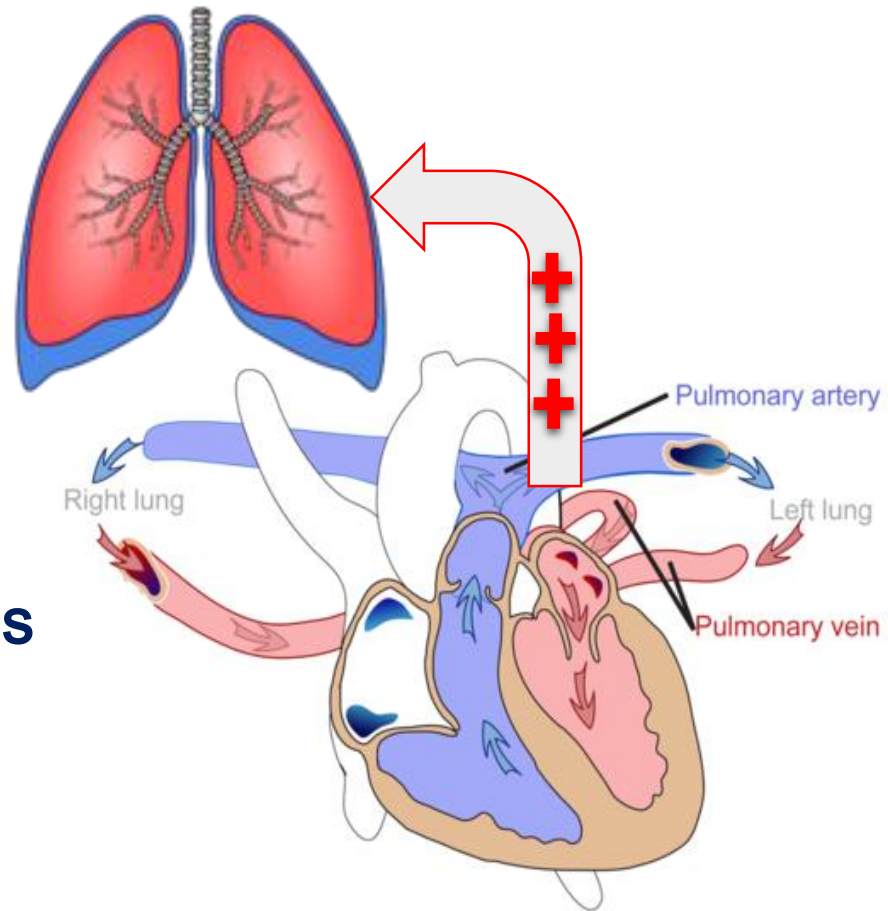
Definition

Accumulation of fluid in the alveoli

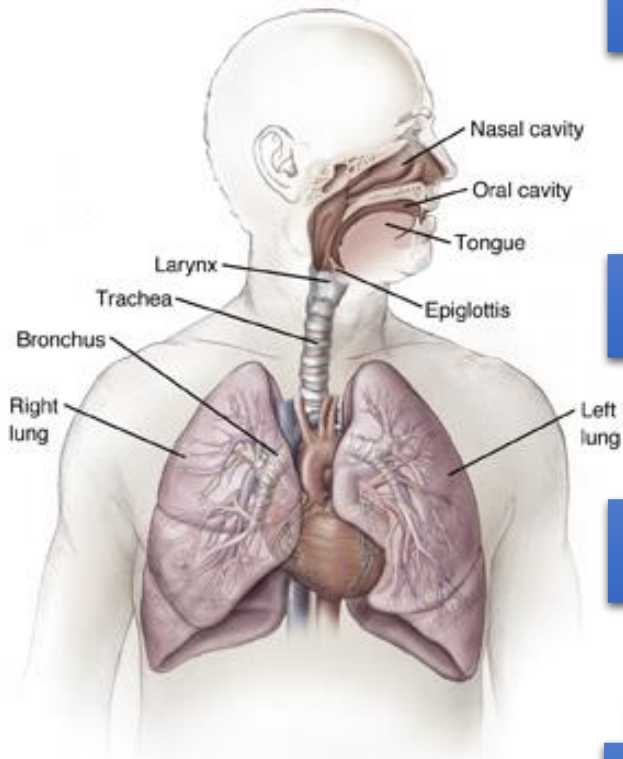


Aetiology

- ① Mitral Stenosis
- ② LSHF
- ③ +++ Lung Infections
- ④ Inhalation of toxins
- ⑤ Snake Venom
- ⑥ Aspiration of gastric acid contents
- ⑦ Acute Hemorrhagic pancreatitis
- ⑧ Narcotic overdose
- ⑨ Pulmonary embolism
- ⑩ Eclampsia



Clinical Picture



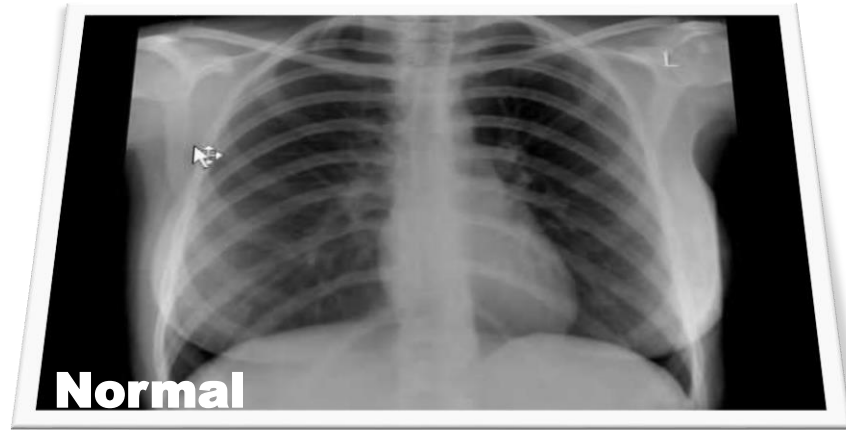
Hypoxemia may cause:
Anxiety- Excessive Sweating- and +++ Dyspnea

Expectoration Of Frothy blood tinged sputum

Fine crackles on Chest Auscultation

Respiratory Failure (CO₂ + Acidosis)

Special Investigations

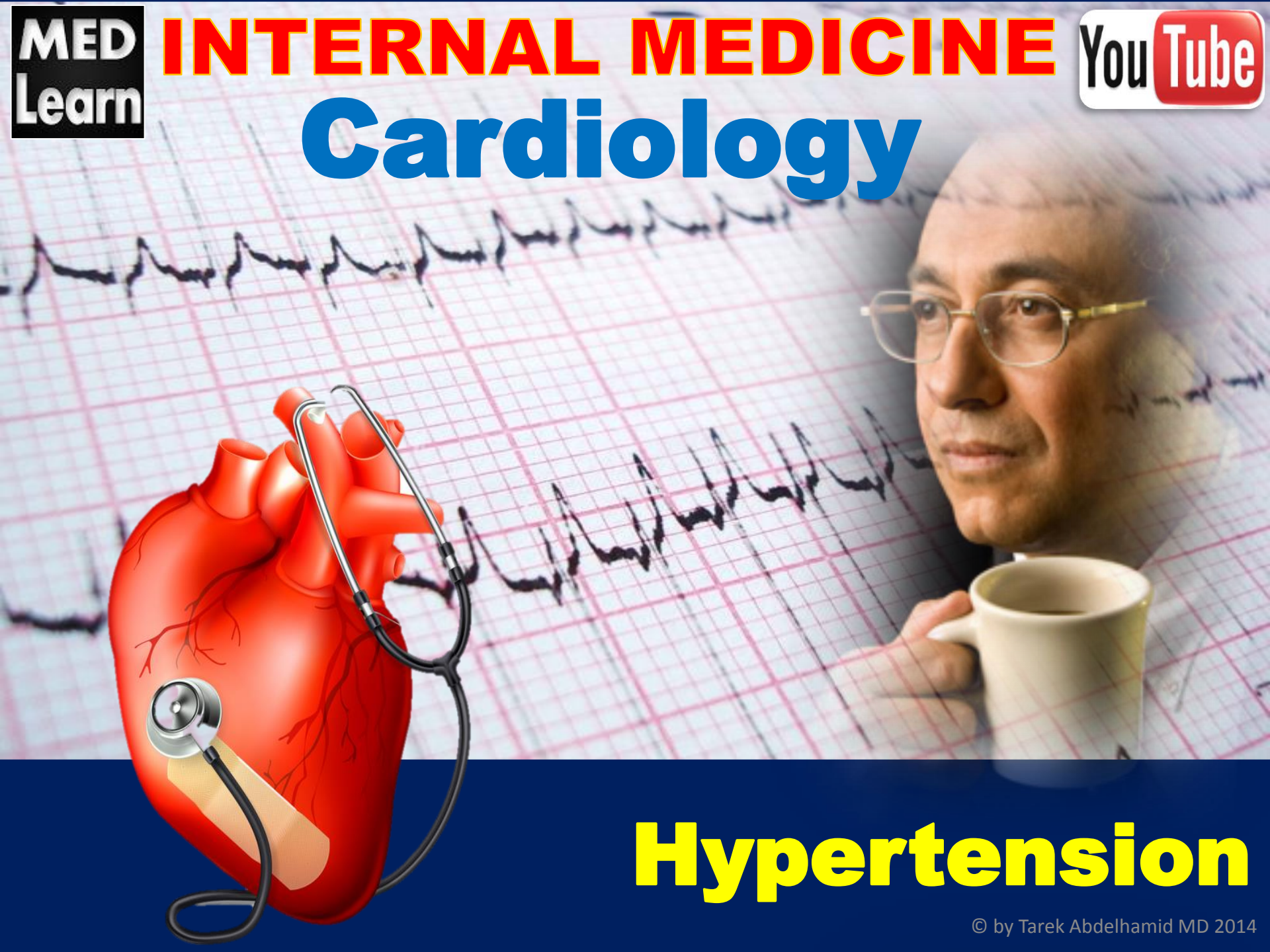


Treatment

- 1- Morphine (5 mg Repeated)
 - 2- 100% Oxygen Inhalation
 - 3- Semi sitting position
 - 4- IV Loop Diuretics e.g. Furosemide 100 mg
 - 5- Afterload reduction by Vasodilators (e.g. Na Nitroprusside IV Drip 25 micro gram/ min)
- Note: Can be used ONLY if Systolic BP is More than 100 mmHg.
- 6- Inotropic drugs for patients with associated HF e.g. Digoxin – Dopamine - Dobutamine
 - 7- Aminophylline (250 mg IV very slowly) Decreases bronchospasm- increases RBF- Increases Na Excretion- Increases Myocardial Contractility
 - 8- If all measures failed you may use: Rotatory tourniquet method.



Cardiology



Hypertension

تحذير هام

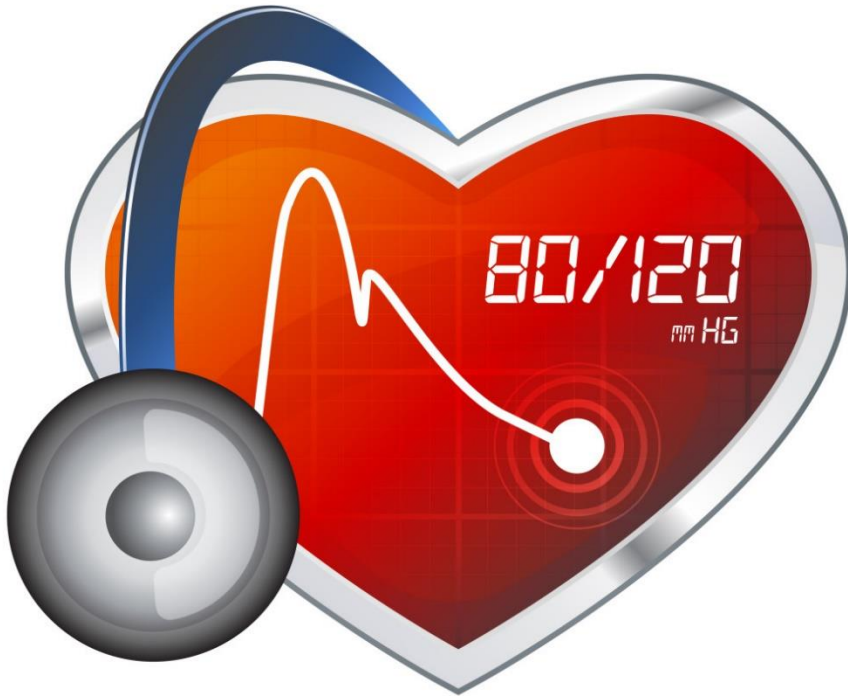
هذا الفيديو و جميع الرسوم التوضيحية المتاحة فى هذا الموقع هم تحت حماية قانون الملكية الفكرية

هذا الفيديو و جميع الرسوم التوضيحية المتاحة فى هذا الموقع هم تحت حماية قانون الملكية الفكرية و لا يحق لأحد إستخدامه أو إستنساخه أو إعادة طبعه أو طبع أجزاء منه بدون إذن مسبق من الدكتور طارق عبد الحميد (أو من ينوب عنه قانونياً). و من يتعدى على الملكية الفكرية المذكورة فسوف يتعرض للمسائلة القضائية عن جريمة إنتهاك حقوق الملكية الفكرية. و إستخدام الفيديوهات و المادة العلمية المذكورة مكفول فقط لمشتري واحد فقط و لأخذ تصريح إستخدام لأكثر من شخص برجاء الإتصال بالدكتور طارق عبد الحميد



Definition

Persistent elevation of diastolic BP More than 90 mmHg (Note: BP should be measured 3 times while the patient is relaxed)



Aetiology (1)

Primary Hypertension (Essential or Idiopathic)

Mild: 90-104

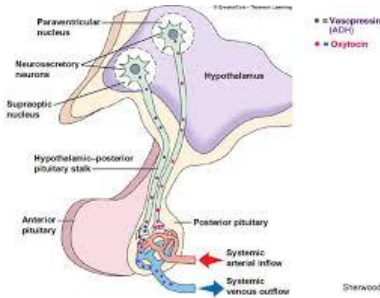
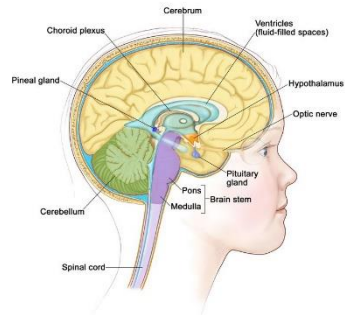
Moderate: 105- 114

Severe: More than 115

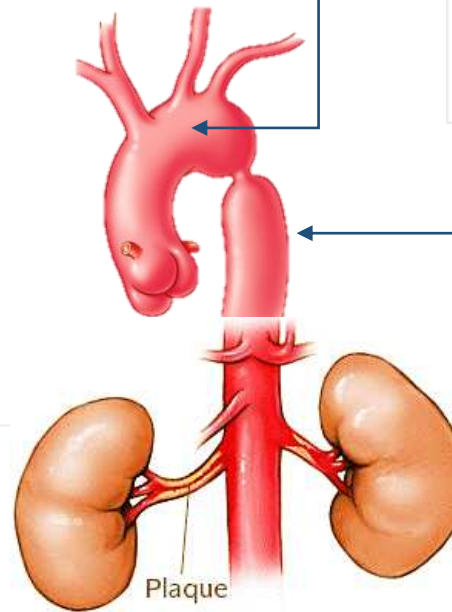
**Malignant: Associated
with end organ damage**

Aetiology (2)

Secondary Hypertension



- 1- Acromegaly
- 2- Pheochromocytoma
- 3- Cushing Syndrome and Conn's syndrome



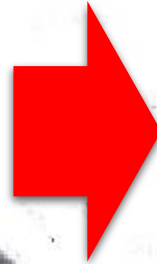
- 1- Contraceptive Pills
- 2- Eclampsia
- 3- Acute Intermittent Porphyrria

- 1- PRV
- 2- Coarctation of the Aorta
- 3- RAS

- 1- AGN
- 2- PCK
- 3- CPN

Clinical Picture

- 1- Asymptomatic
- 2- Headache
- 3- Dizziness
- 4- Tinnitus
- 5- Epistaxis

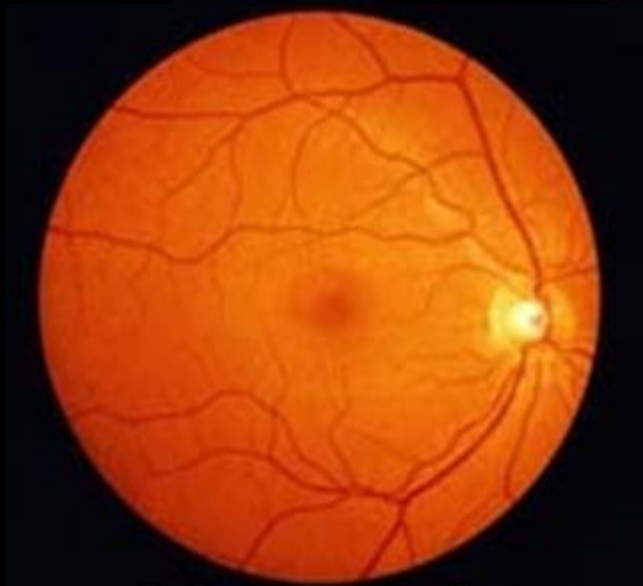


Encephalopathy
Retinopathy
Heart Failure
Renal Failure

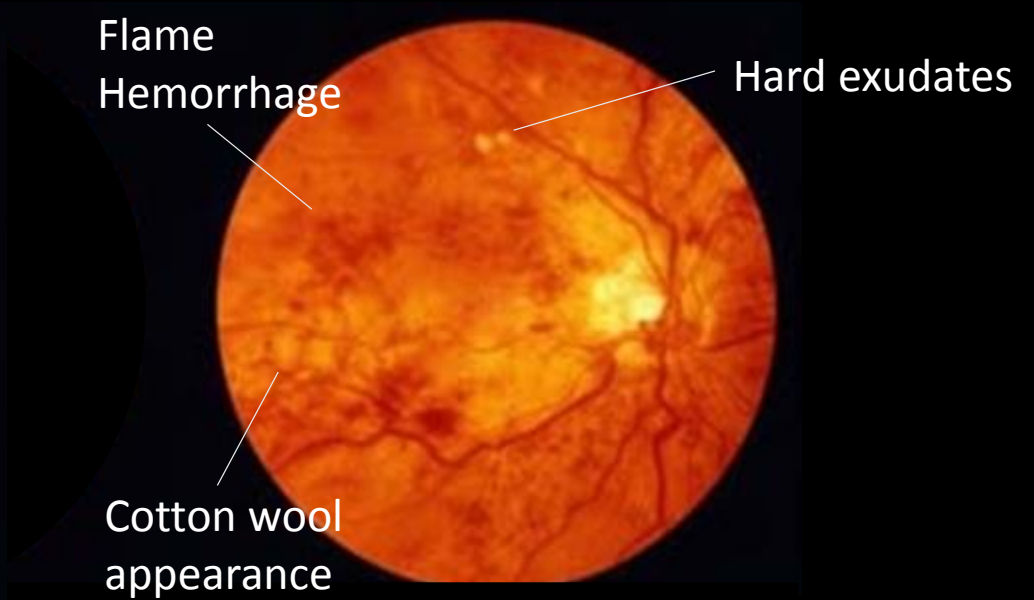


Special Investigations

- ① Measure the Blood Pressure
- ② Investigations to exclude secondary causes
- ③ Ophthalmoscopy
- ④ X-Ray to detect LVH (Note: the existence of LVH indicated long standing condition)
- ⑤ Renal Function Tests



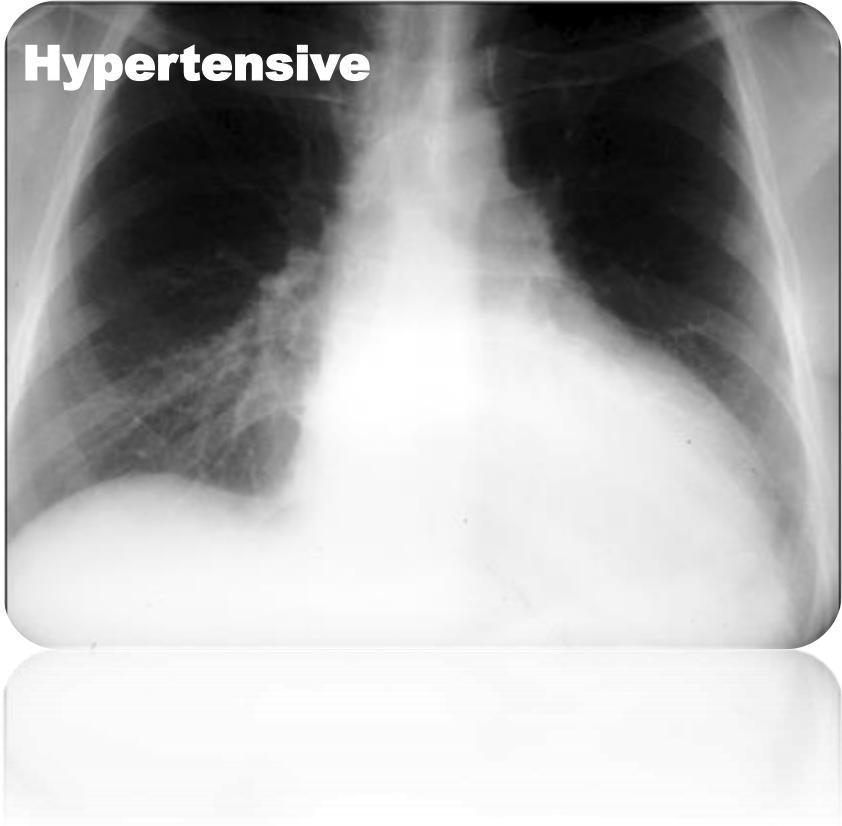
Normal Retina



Hypertensive Retinopathy

LVH + ↑ BP

Long Standing



Treatment

Non Pharmacological treatment

- 1 Decrease Na intake
- 2 Diet rich in fruits, vegetables, and low in saturated fat
- 3 Stop smoking
- 4 Decrease alcohol intake
- 5 Increase aerobic exercise
- 6 Diet rich in Ca, Mg, K (may be helpful)

Hypertension 2014

Note: Beetroot Juice
Lowers Blood
Pressure in
Hypertensives

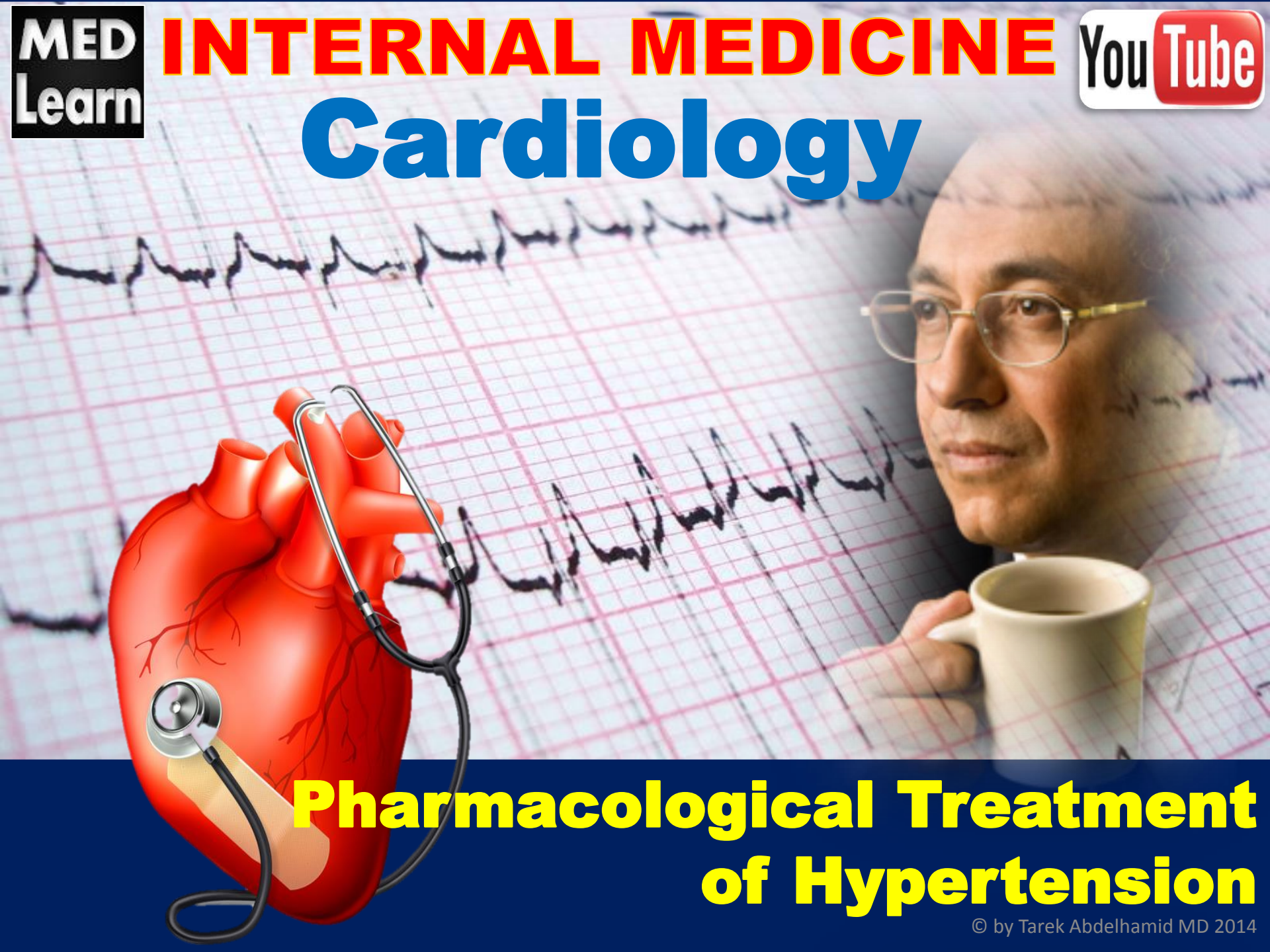
Treatment

Pharmacological treatment

ACE- I
Aldosterone Antagonists
Alpha Blockers
Beta Blockers
Ca-Channel Blockers
Sympatholytics
Diuretics



Cardiology

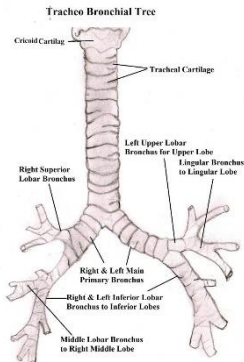


Pharmacological Treatment of Hypertension

Important Information



Beta Blockers



May induce
Bronchospasm (CI in
Bronchial Asthma and
COPD)

Thiazide Diuretics

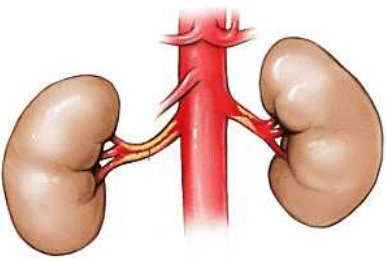
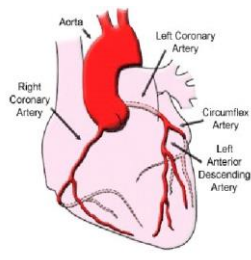


Hypokalemia-Increase
Uric Acid and
Increase Glucose

Aldosterone Antagonists



Hyperkalemia (Avoid
them in patients with
Renal insufficiency or
Renal Failure)

ACE-I  CI in bilateral RAS	① May Precipitate ARF in patients with Bilateral RAS ② May cause Cough as troublesome side effect ③ Angioedema may develop secondary to inhibition of Kininase
Ca-Channel Blockers 	May inhibit myocardial contractility and thus may precipitate Heart Failure
Alpha Blockers 	May cause Marked Hypotension and Syncope (give small dose at bed time)

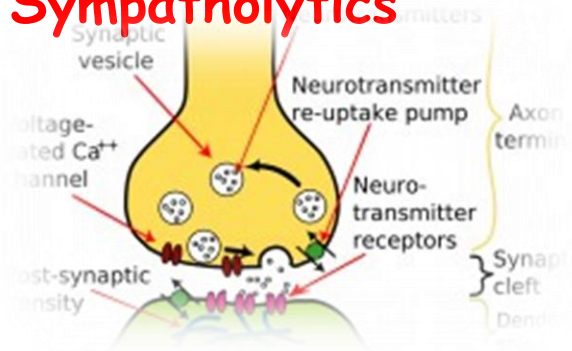
Central Sympatholytics:



Methyldopa

Associated with high frequency of drug intolerance such as fatigue- Sedation- Postural Hypotension- and Impotence.

Peripheral Sympatholytics

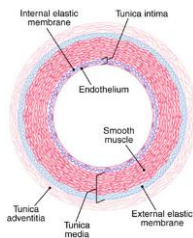


Reserpine: (Depression- Nasal stuffiness- Peptic Ulcer)

Guanethidine:

- 1- Postural hypotension
- 2- # TCAD
- 3- May cause hypertensive crisis in patients with Pheochromocytoma

Arteriolar Vasodilators: Hydralazine

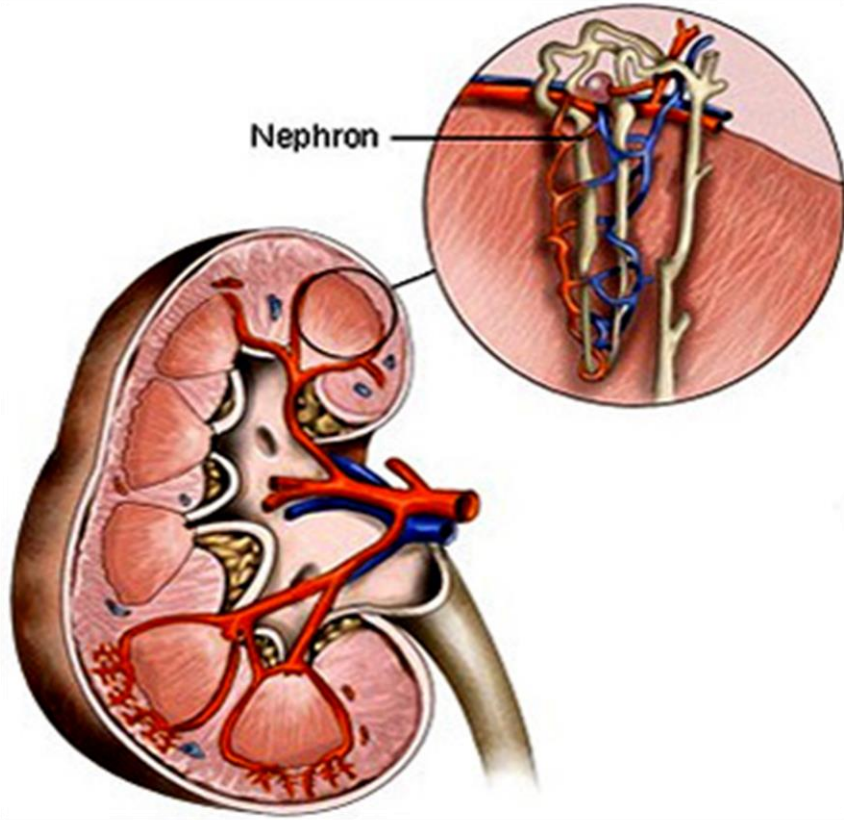


May cause reflex tachycardia and thus ppt Angina! (Avoid in CHD)

Choosing Antihypertensive Medication

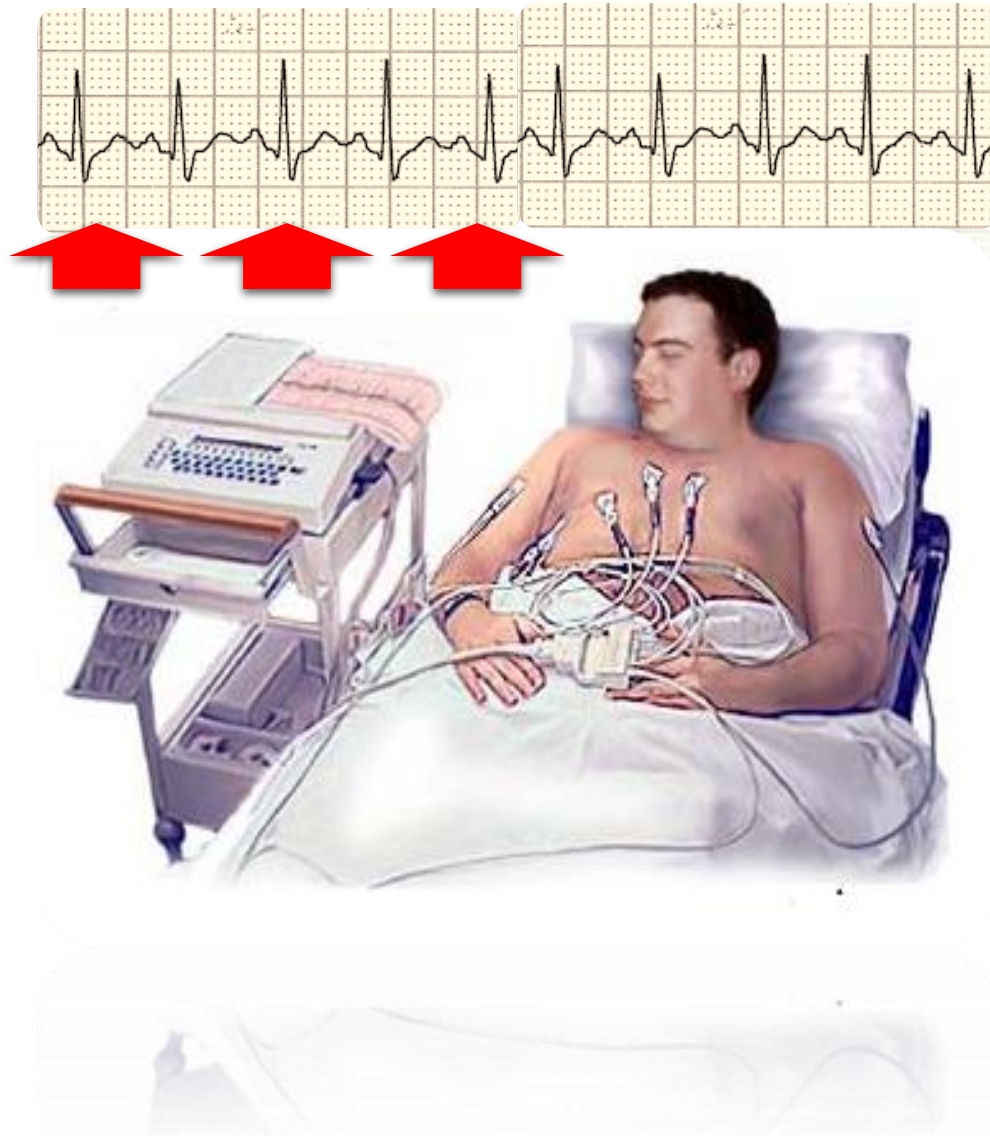


Diabetic Nephropathy



ACE-I

Heart Failure



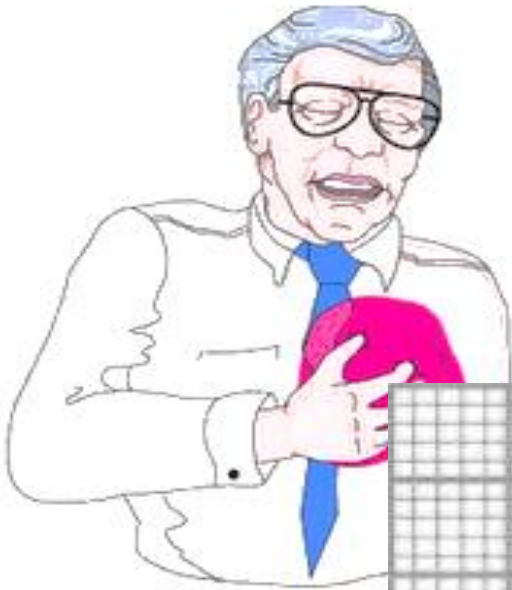
**ACE-I OR/
Diuretics**

Tachyarrhythmias

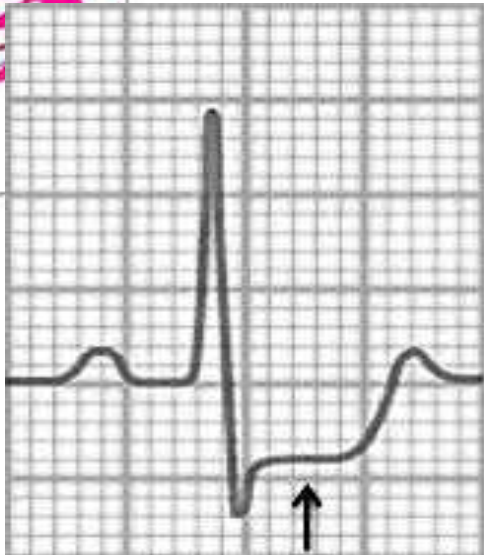


**Beta
Blockers**

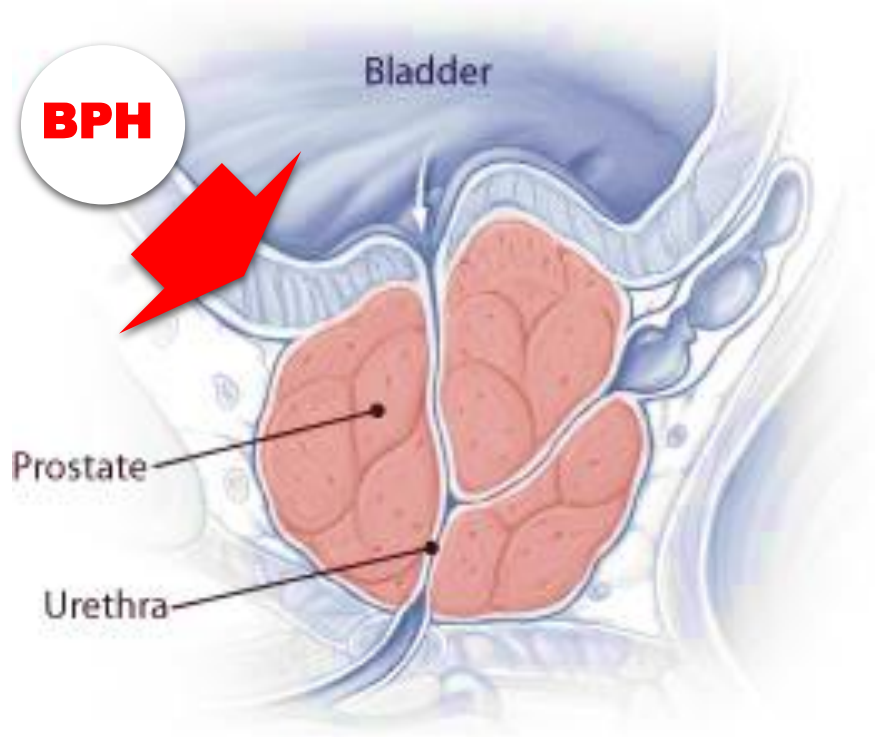
Angina



**Beta Blockers
or Ca Channel
Blockers**



Prostatic Hypertrophy



**Alpha
Blockers**

Pregnancy



**Alpha
Methyldopa or
Hydralazine**



Avoid

Gout



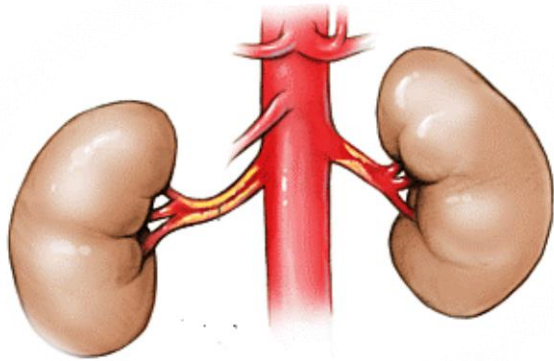
Diuretics

Bronchial Asthma



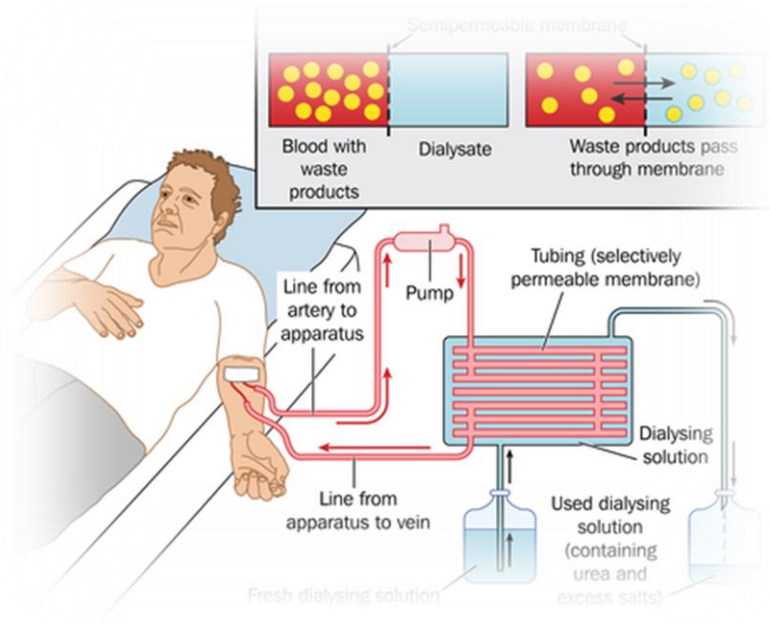
**Beta
Blockers**

Renal Artery Stenosis



ACE-I

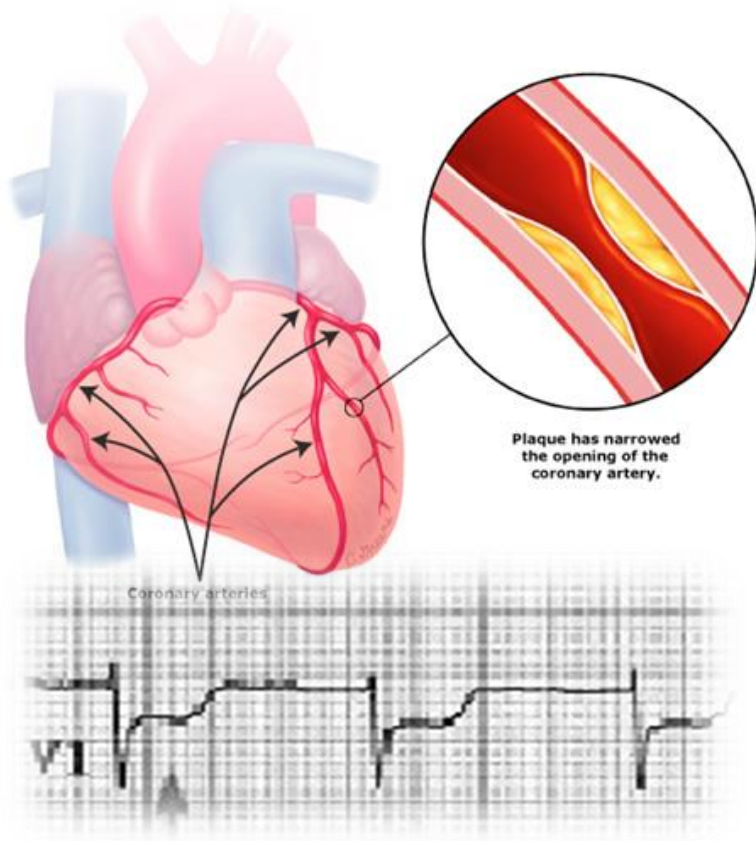
Renal Failure



Avoid

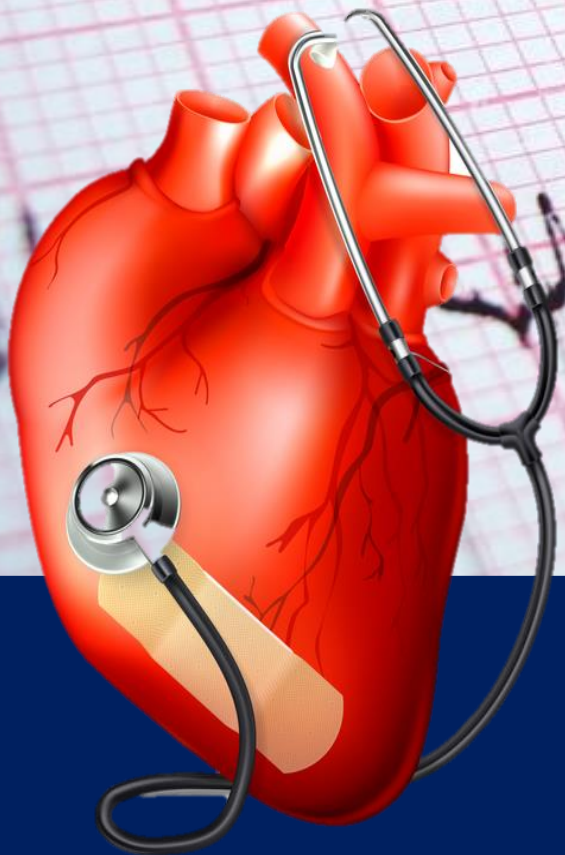
**Aldosterone
Antagonists**

Angina



Avoid → Hydralazine

Cardiology



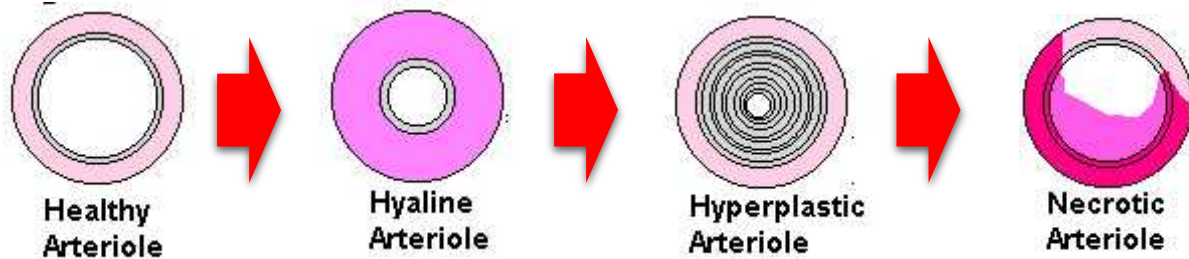
Hypertensive Crisis

Definition

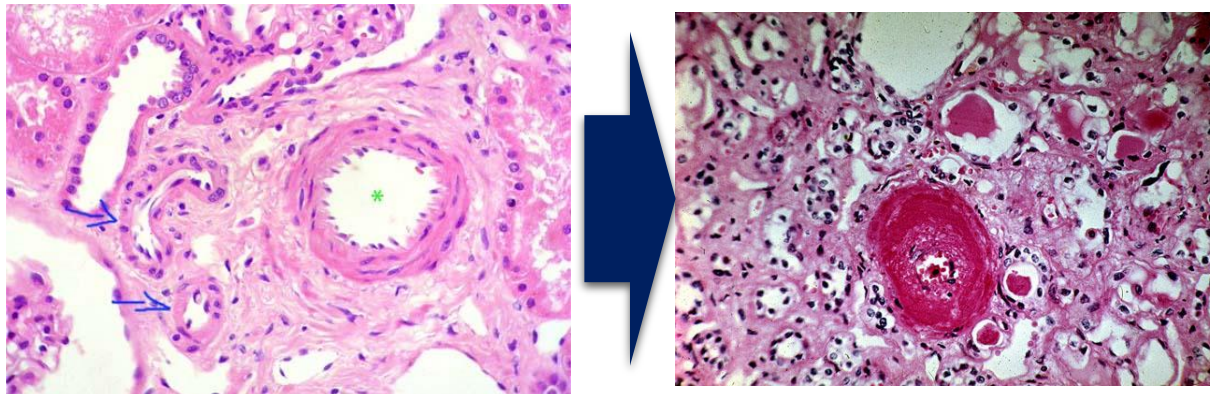
A life threatening Medical Emergency characterized by marked elevation of BP associated with features of end organ damage



Mechanism



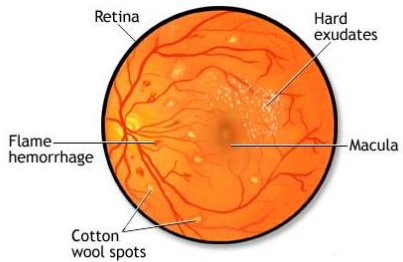
Fibrinoid necrosis of small sized arteries and arterioles



Complications



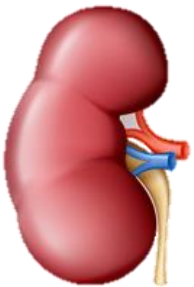
Encephalopathy



Retinopathy



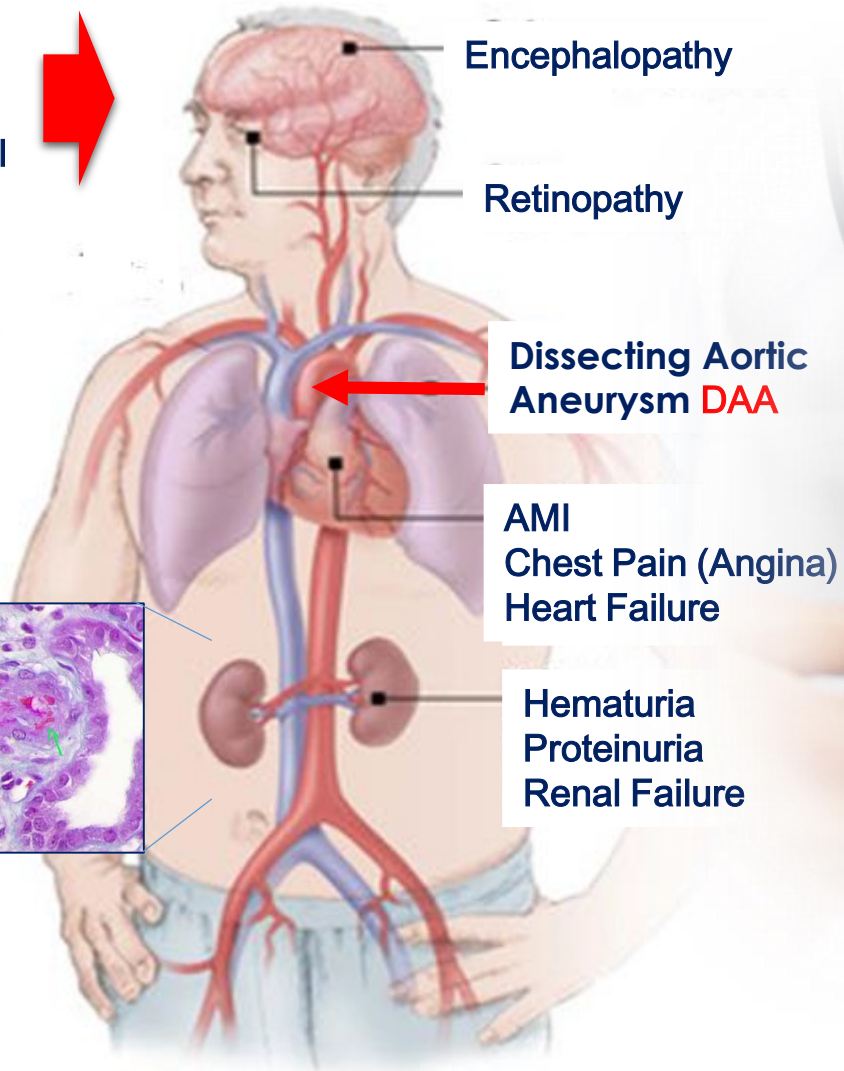
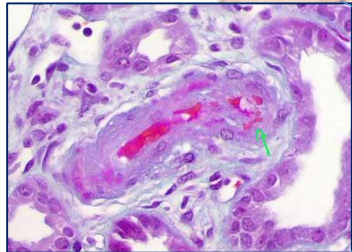
Heart Failure



Renal Failure

Clinical Manifestations

Headache
Irritability
Confusion
Altered Mental
status



**Diastolic BP More
than 130 mmHg**

≥



Treatment

The aim is to Decrease the elevated BP by 25% within 1 hour to protect from end organ damage (Avoid fast lowering of BP as it may precipitate ARF).

① **Na Nitroprusside**

Dilates both arterioles and veins

May cause Acute Cyanide Poisoning (ttx by Amyl Nitrite Inhalation)

② **Fenoldopam (VD)** (Increases RBF) Activating peripheral Dopamine D_1 receptors causing VD of arterioles

③ **Diazoxide (VD)** IV bolus- Mimi bolus dose regimen (Maximum of 150 mg)

Never use it if you suspected Dissecting Aortic Aneurysm or AMI- Increases Blood Glucose Level

④ **Furosemide** (Lasix): Important adjunct therapy as it causes Na-Diuresis and speeds up the recovery from encephalopathy and Retinopathy.

⑤ **Beta Blockers** are recommended for patients with evidence of DAA

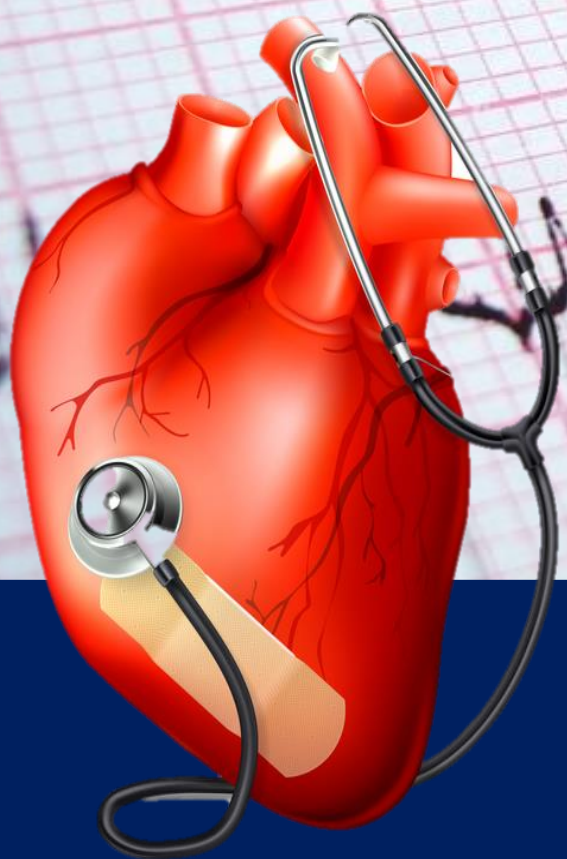
(Chest pain radiating to the Back)

Other medications include:

Nifedipine (Adalat)- Hydralazine- Arfonad



Cardiology



Atrial Premature Contractions

Definition

Occasional ectopic beats originating from the atrium

Aetiology

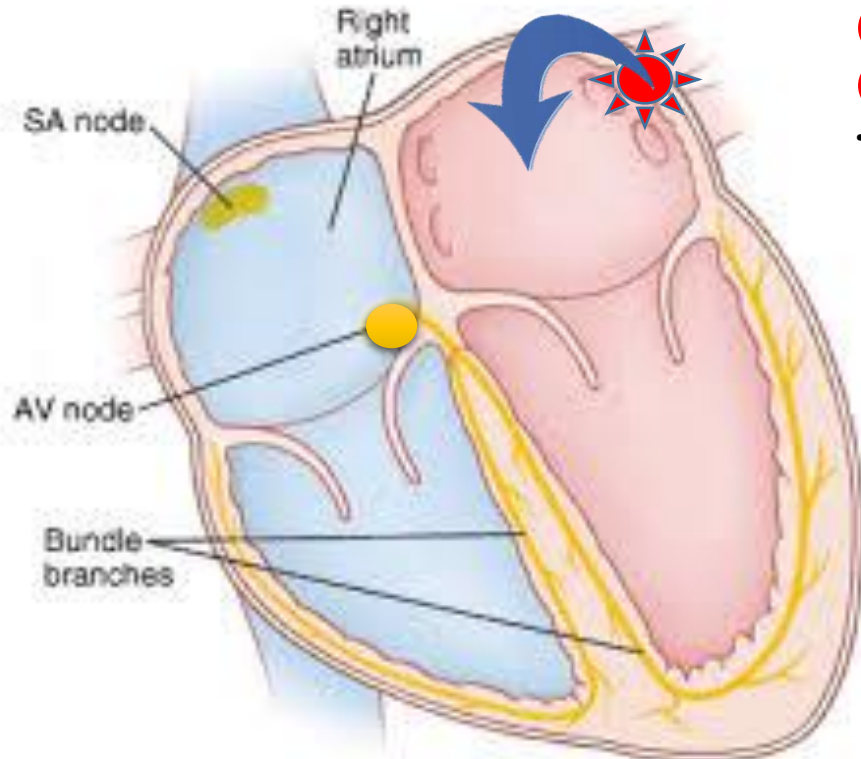
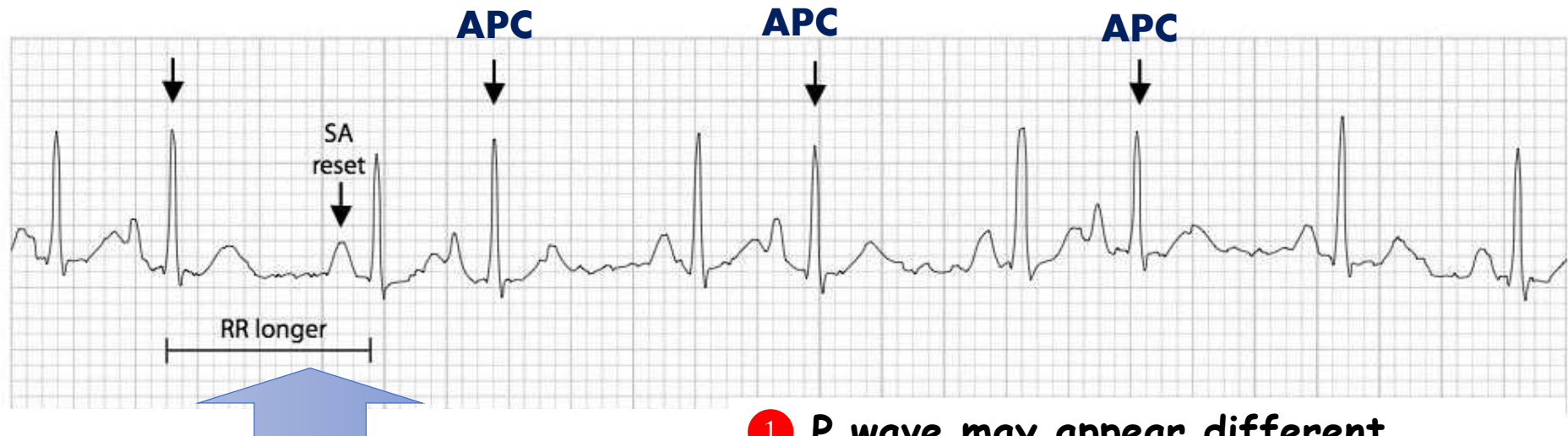
Commonly occurs in a normal heart

Clinical Picture

Palpitations



Special Investigations

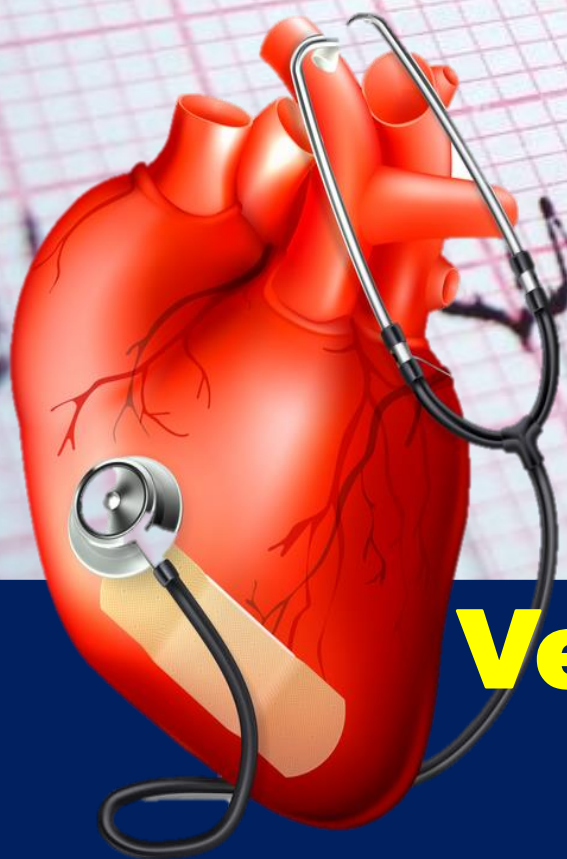


- 1 P wave may appear different
- 2 QRS is typically NORMAL
- 3 If aberrant conduction e.g. RBBB the QRS may be wide

Treatment

Usually, No Medical treatment is needed

Cardiology



Ventricular Premature Contractions

Definition

Ectopic beats originating from a focus in the ventricle

Aetiology

- 1- Electrolyte abnormalities**
- 2- Thyrotoxicosis**
- 3- IHD**
- 4- Digitalis toxicity**

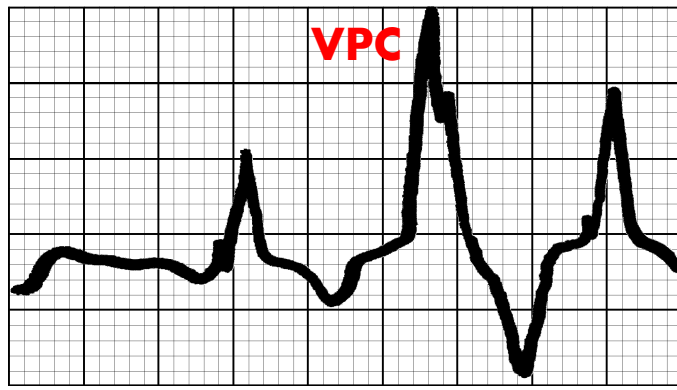
Clinical Picture

Palpitations

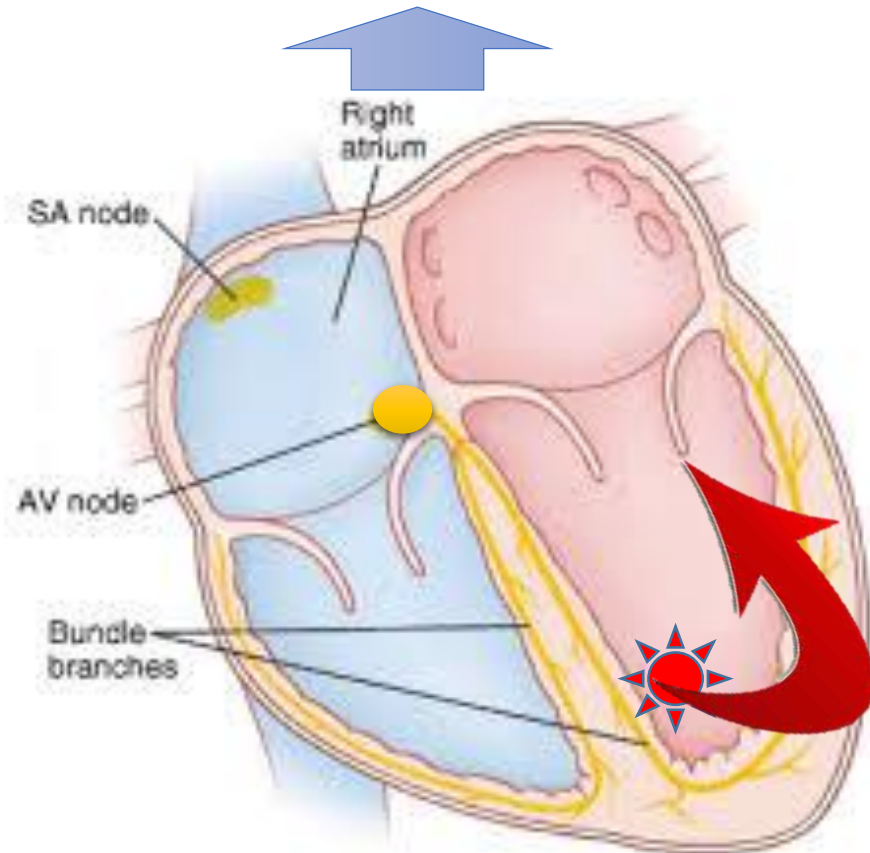


May precipitate Heart Failure and sudden death especially in patients with pre-existing heart disease

Special Investigations



Wide Bizarre QRS Complexes are the most characteristic feature on ECG

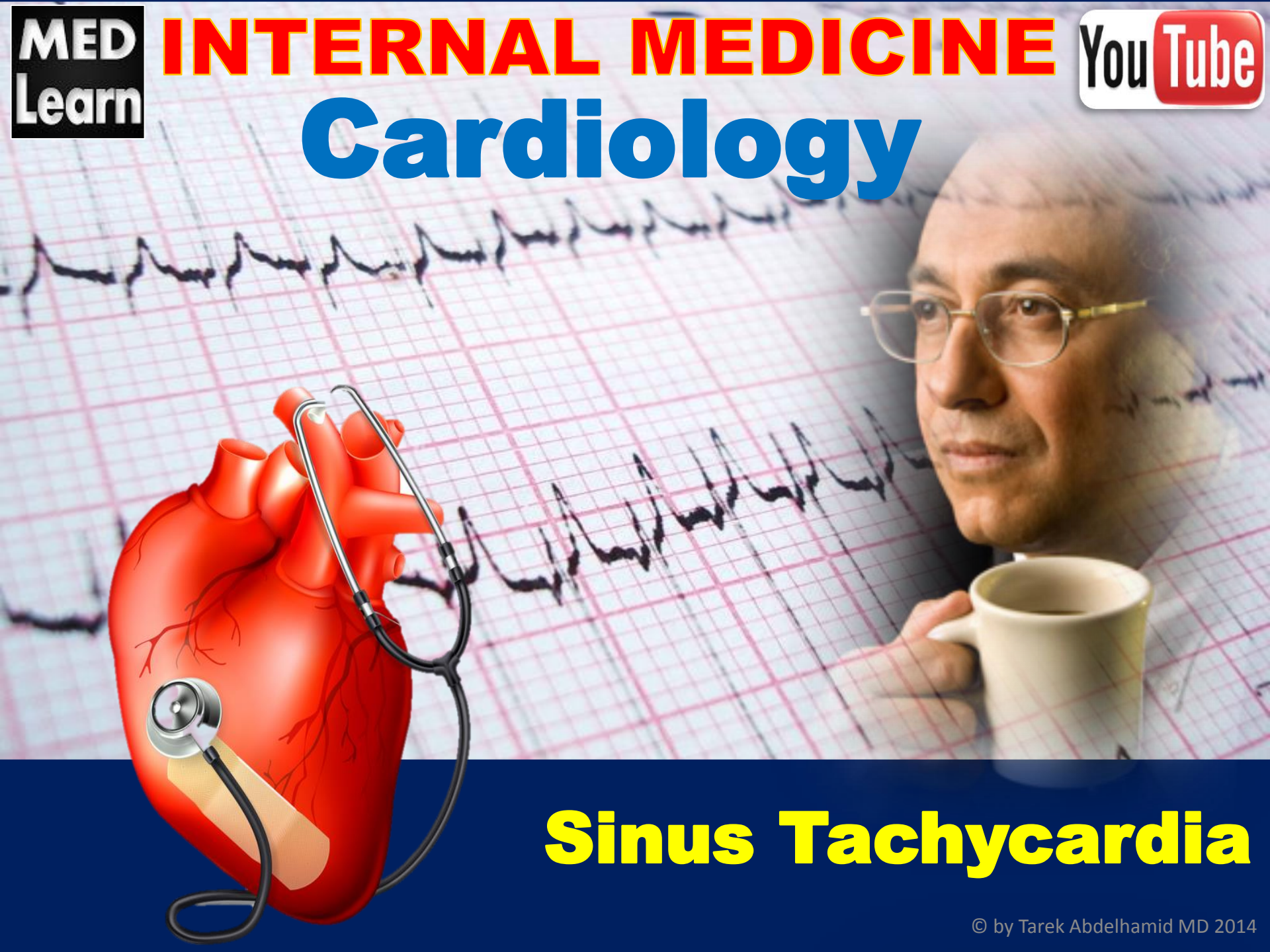


Treatment

- ① If Asymptomatic:
No medical treatment is usually needed
- ② Medical treatment by **Beta Blockers** is indicated only for symptomatic cases
- ③ Treatment of any **underlying causes** is essential



Cardiology



Sinus Tachycardia

Definition

Increase heart rate due to increased automaticity of SAN (Rapid impulse discharge of the SAN)

Aetiology

Typically occurs as a physiological response to:

- ① Anemia
- ② Pain
- ③ CHF
- ④ Fevers
- ⑤ Hyperthyroidism
- ⑥ Shock

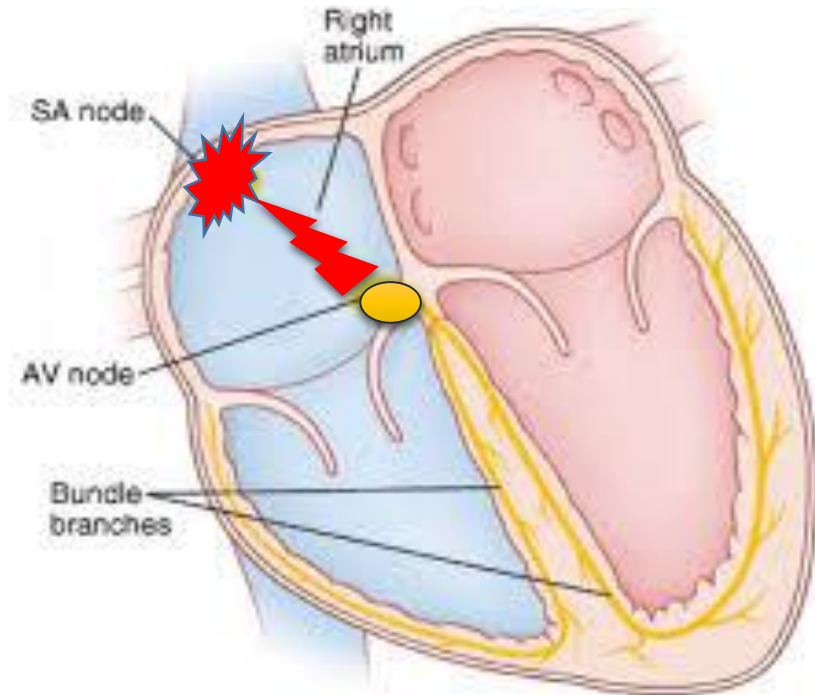
Clinical Picture

The Clinical Picture of the underlying cause is usually apparent

Special Investigations



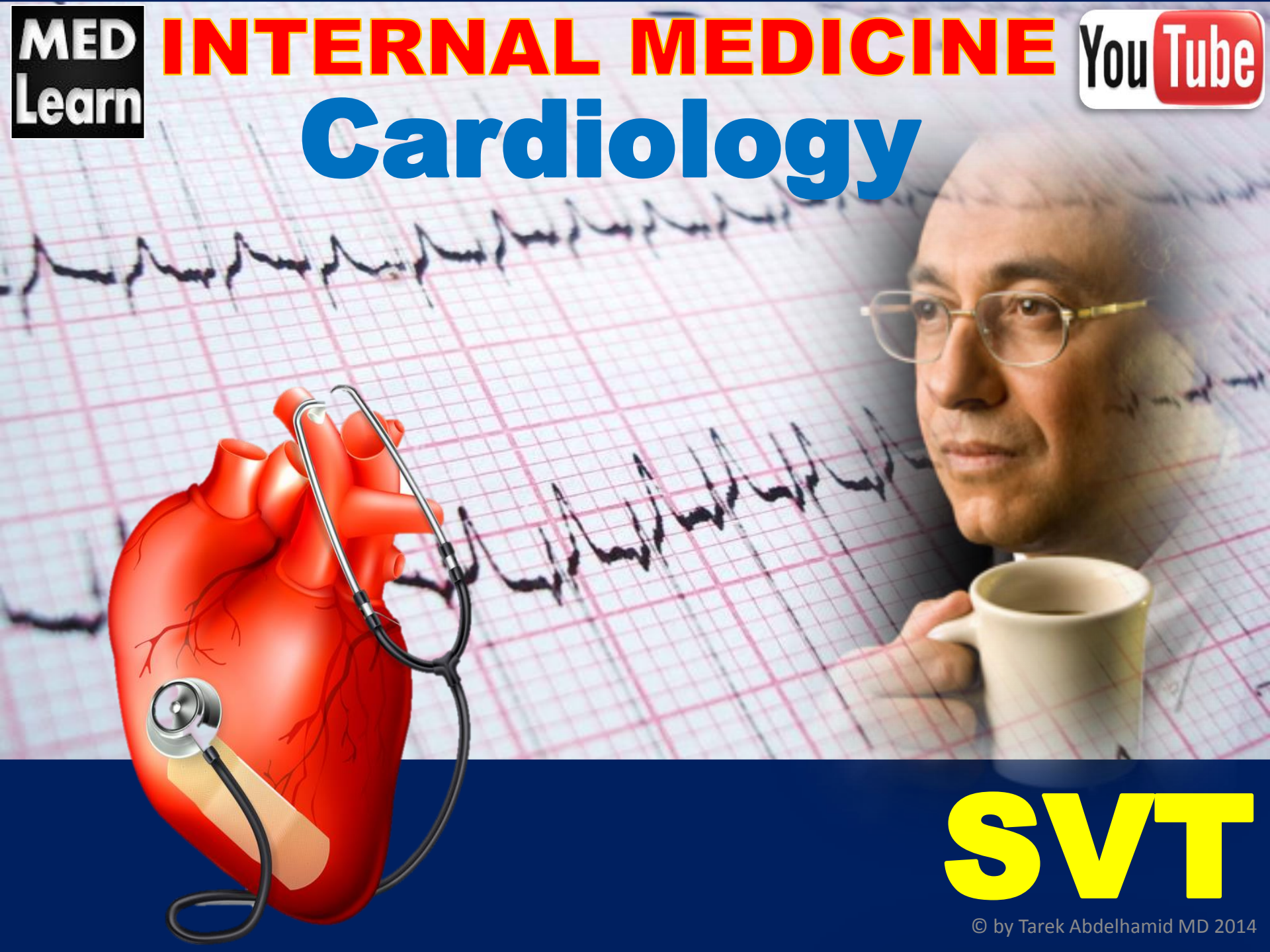
ECG is typically normal with the exception that the HR is more than 100 beats per minute



Treatment

Correct the underlying cause

Cardiology



SVT

Definition

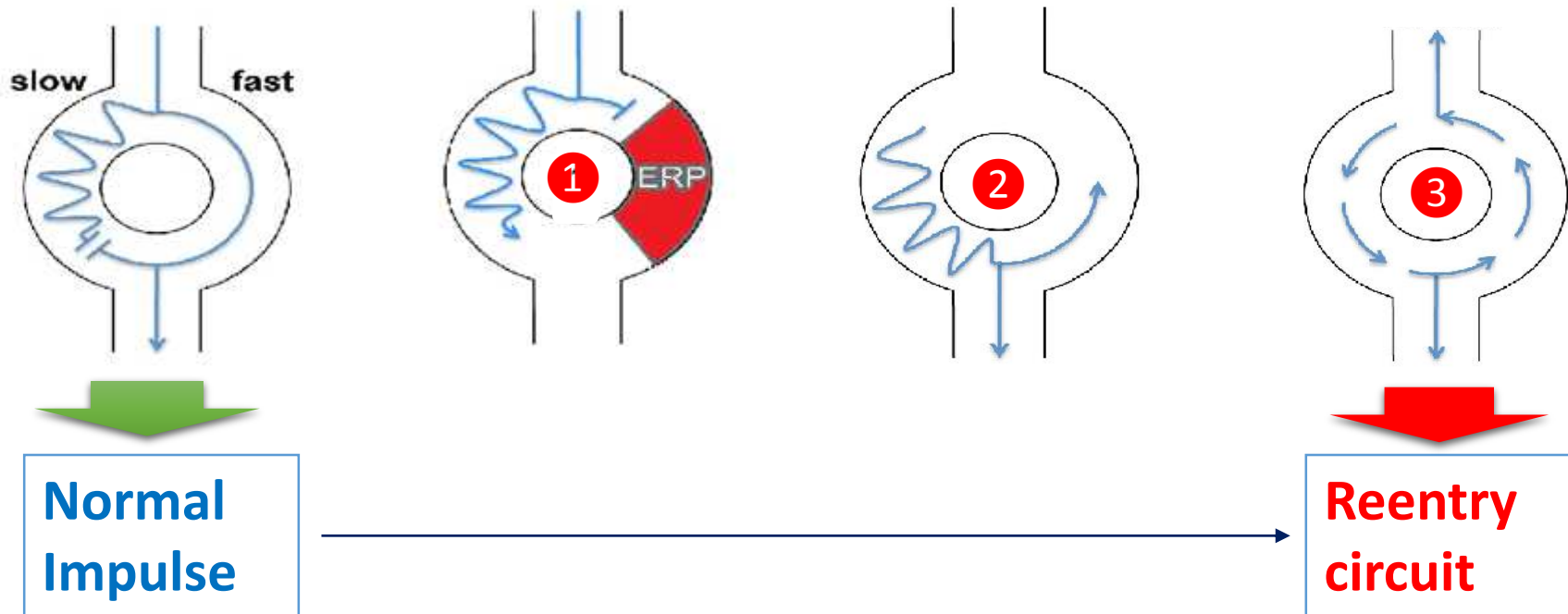
A form of tachycardia caused by an ectopic focus in the atria or AVN (i.e. above the ventricle)

Aetiology

SVT can occur in the following conditions:

- 1- Normal Individuals**
- 2- WPW-S**
- 3- Digitalis +++**
- 4- Hyperthyroidism**
- 5- Organic heart disease**

Mechanism



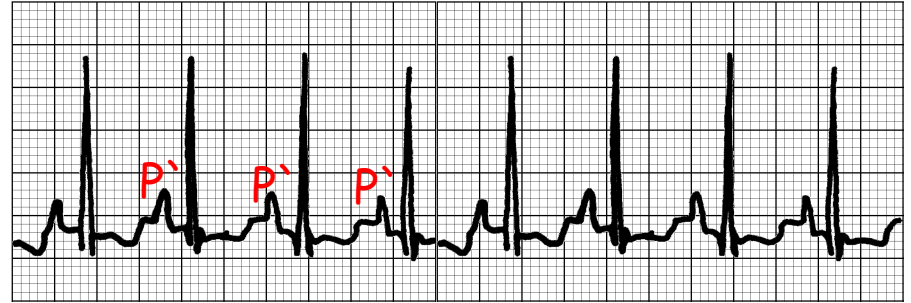
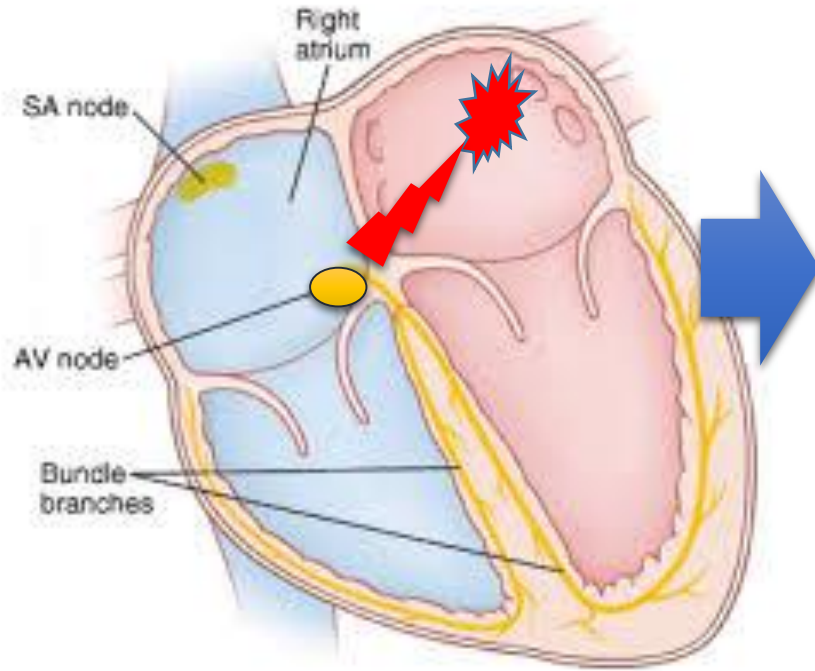
Clinical Picture

Palpitations



May precipitate Heart Failure – Syncope- and Anginal pains especially in patients with underlying heart disease

Special Investigations



QRS Shape is usually normal except if aberrant conduction.

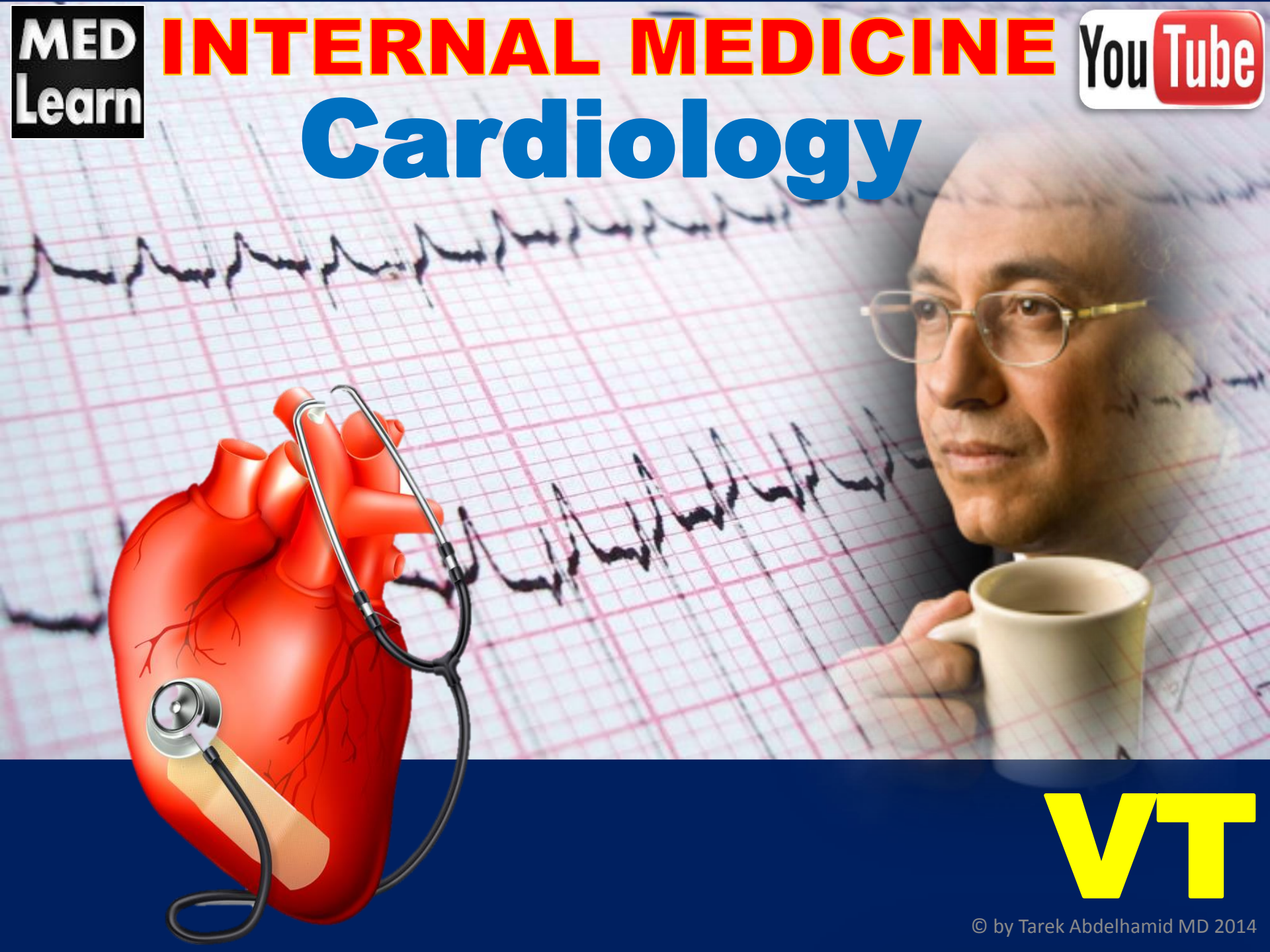
Treatment

- ① Vagal Stimulation e.g. Valsalva maneuver – Stretch of arms- Breath holding- Carotid Sinus massage
- ② **IV Adenosine is the drug of choice** (if vagal stimulation failed)
- ③ Radiofrequency ablation (the procedure of choice in cases of WPW-S)



Give peripherally by rapid bolus either directly into vein or through IV line (followed by saline flush) over 1–2 seconds. Initially 6mg; if no result within 1–2 mins, may give 12mg; may repeat a second 12mg dose if needed. Max 12mg/dose.

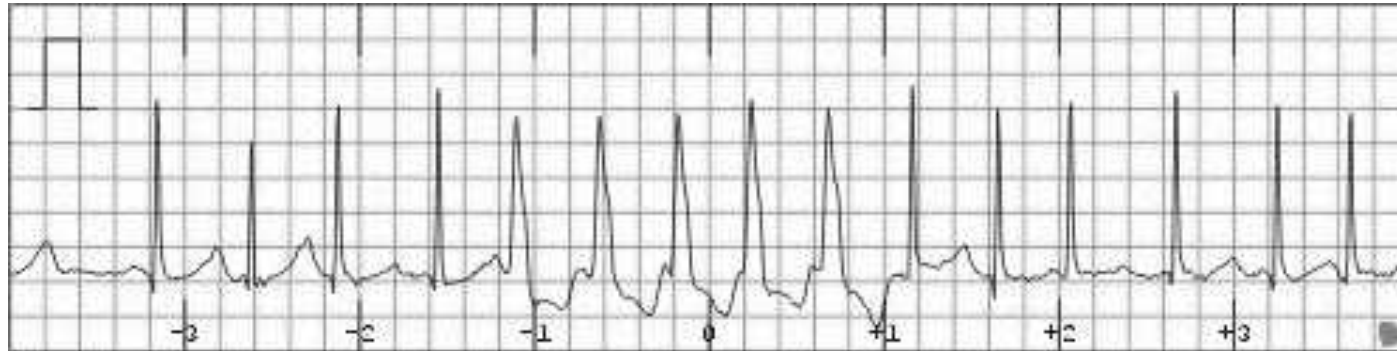
Cardiology



VT

Definition

VT is defined as 3 or More consecutive VPCs



Normal
Rhythm



VT

Aetiology

1- IHD

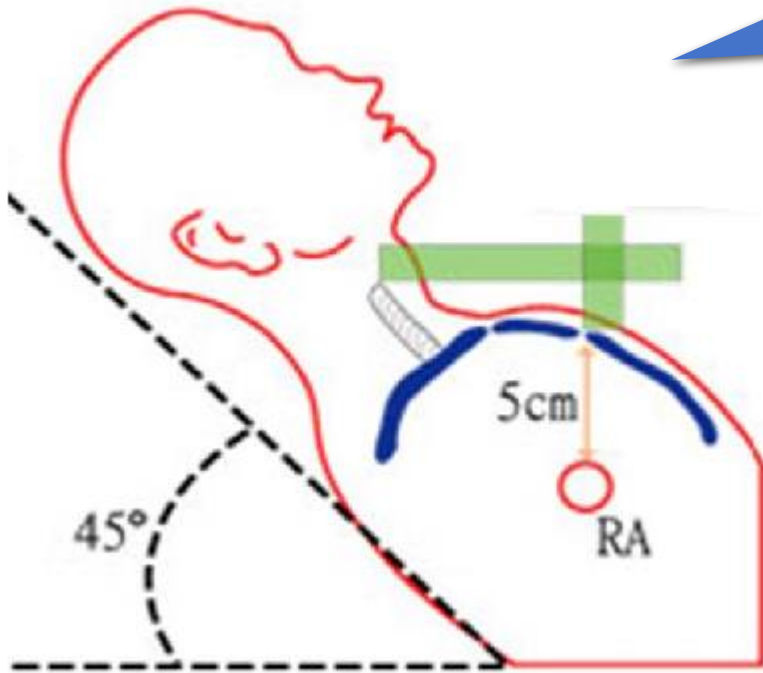
2- Cardiomyopathy

3- Myocarditis

4- Rheumatic heart disease

Clinical Picture

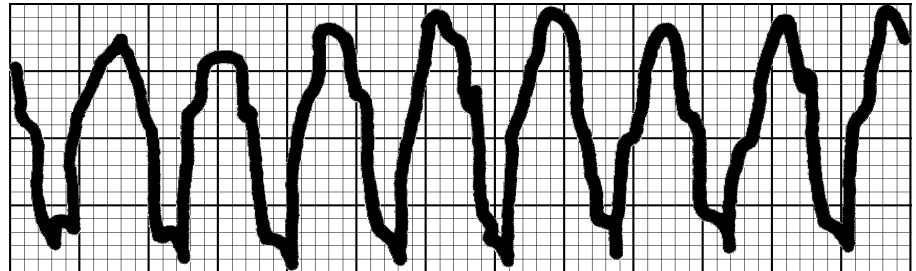
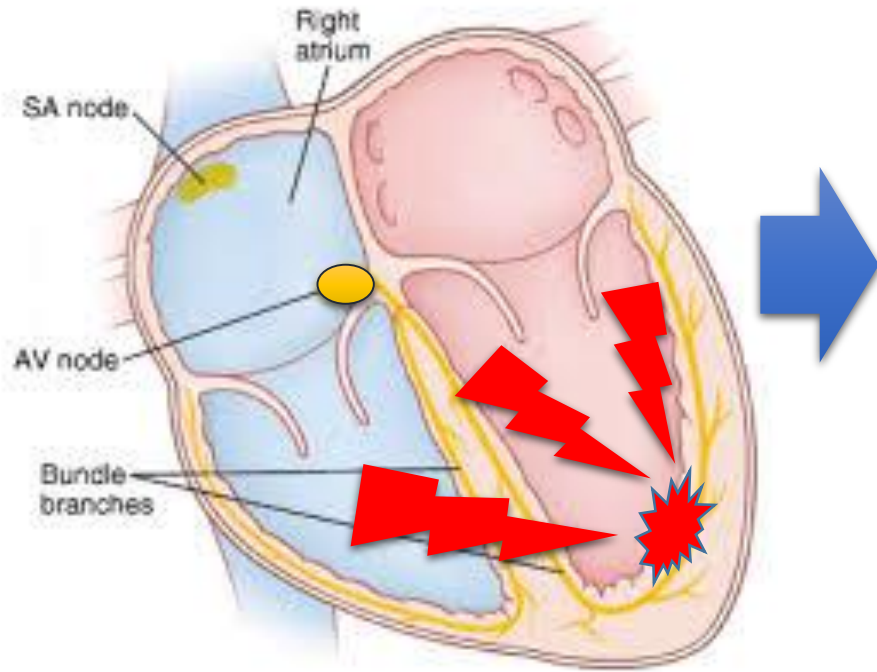
Palpitations



**Frequent Cannon Waves
on JVP**

**May precipitate
Hypotension- Heart
Failure –
Myocardial
ischemia- and
sudden death**

Special Investigations



- ① **Wide bizarre QRS Complexes**
- ② **No relation between P and QRS**
- ③ **HR: 160- 240 beats/ minute**

Note: Sustained if more than 30 seconds in duration

Treatment

① If **complicated** with Hypotension-Heart Failure – Myocardial ischemia

Synchronized DC Conversion
(**Immediately**)

② If the patient is tolerating the VT

Lidocaine

③ Preventing other attacks in cases of Recurrent VT

If Heart Failure

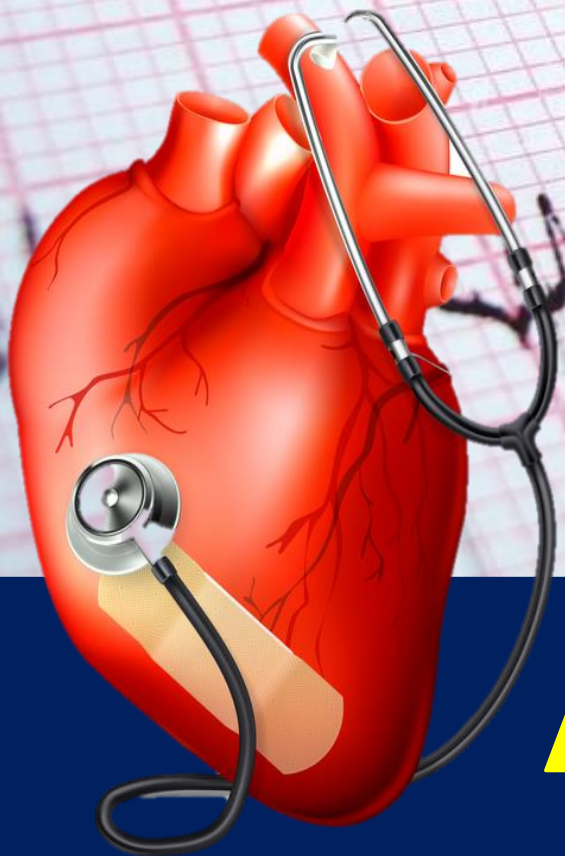
Present

AICD

Absent

Amiodarone
Plus a Beta
Blocker

Cardiology



Atrial Fibrillation

Definition

Atrial Fibrillation (AF) is a form of arrhythmias characterized by rapid, disorganized, and asynchronous atrial contractions

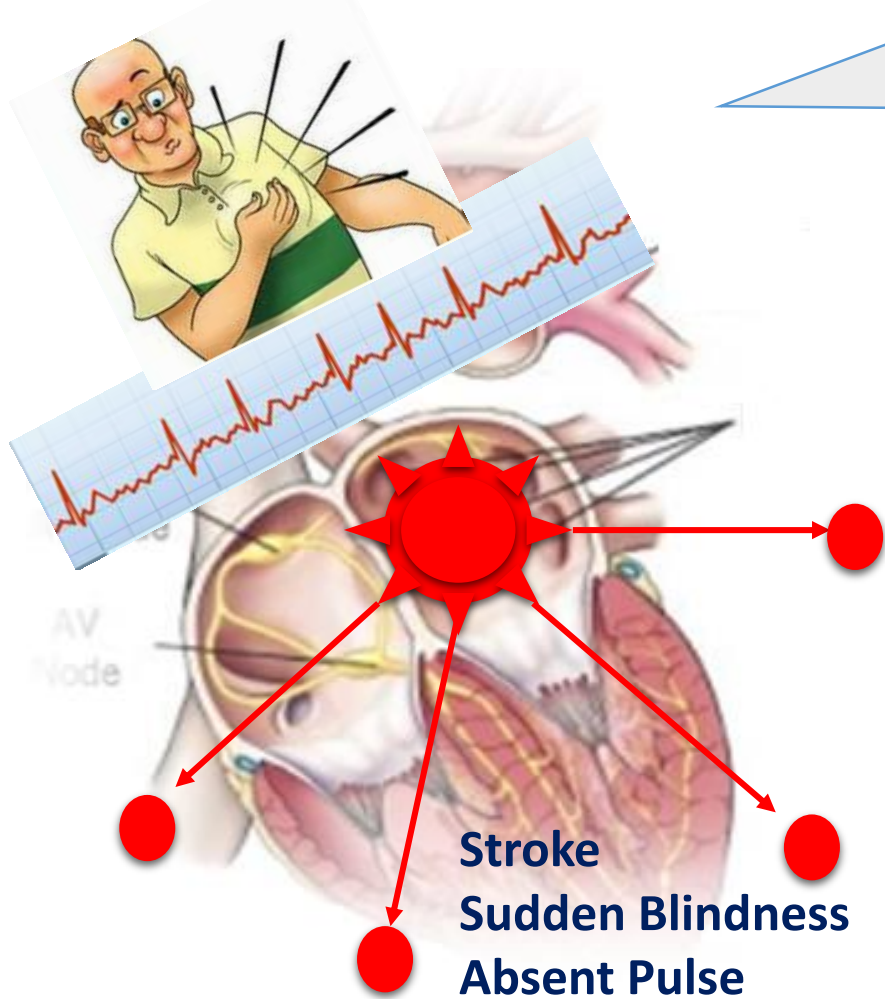
Aetiology

Causes of AF include:

- 1) Idiopathic
- 2) Hypertension
- 3) Ischemic Heart Disease (IHD)
- 4) Mitral Stenosis
- 5) Constrictive Pericarditis
- 6) T3++++, Digitalis toxicity, myocarditis
- 7) Cardiomyopathy

Clinical Picture

Irregular Palpitations

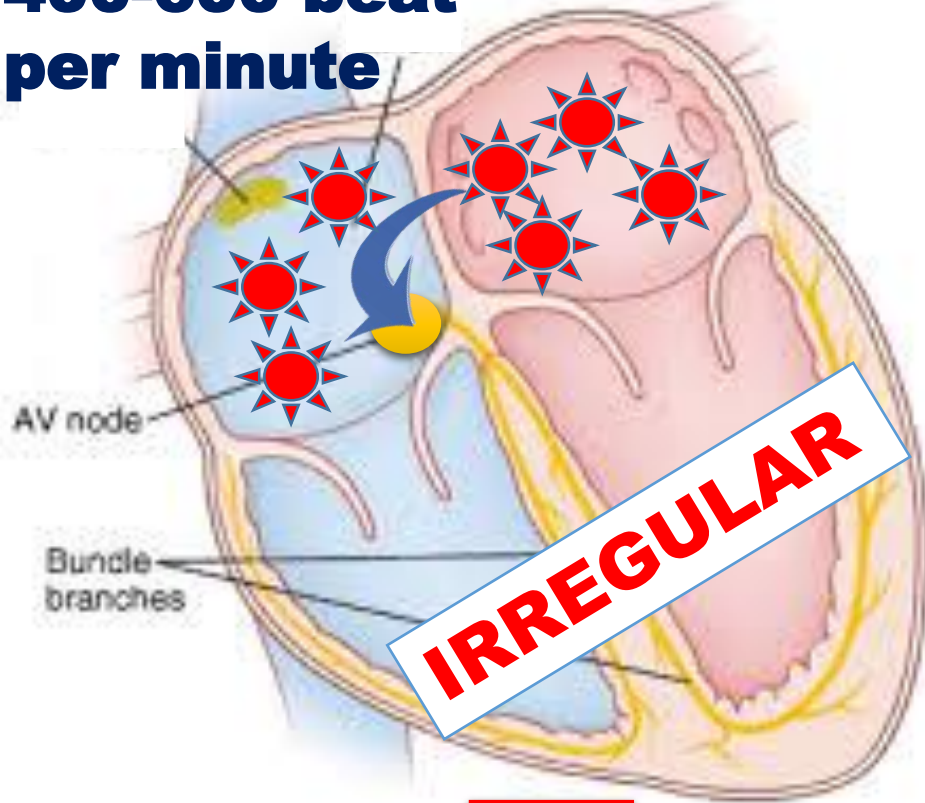


**May precipitate APO–
Angina- Syncope
especially in patients
with underlying heart
disease**

Embolization

Special Investigations

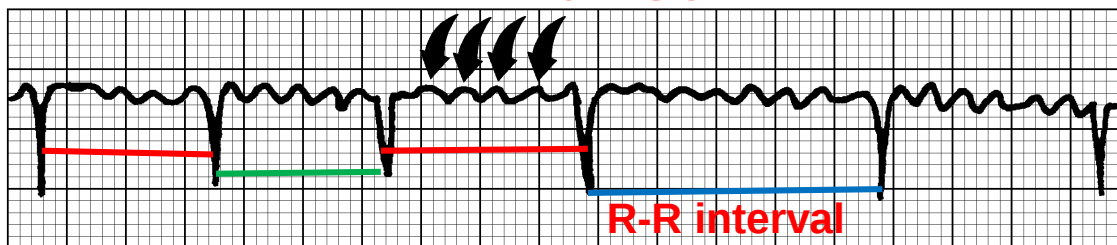
**400-600 beat
per minute**



- ① Totally irregular cardiac rhythm
- ② Absent P waves
- ③ The base line of the heart shows fibrillatory “f” waves (instead of the P waves)



“f” waves



Treatment



① Start anticoagulation to protect from Stroke

② Cardioversion by DC Shock or Quinidine

Cardioversion should not be done unless the patient is anticoagulated for at least 3 weeks.

③ Immediate cardioversion

(without waiting for the 3 Weeks) is indicated in the following conditions (Angina- CHF- Associated with WPW-S with rapid ventricular response)

Cardioversion is generally NOT recommended if AF More than 48 hours.....Embolization?!

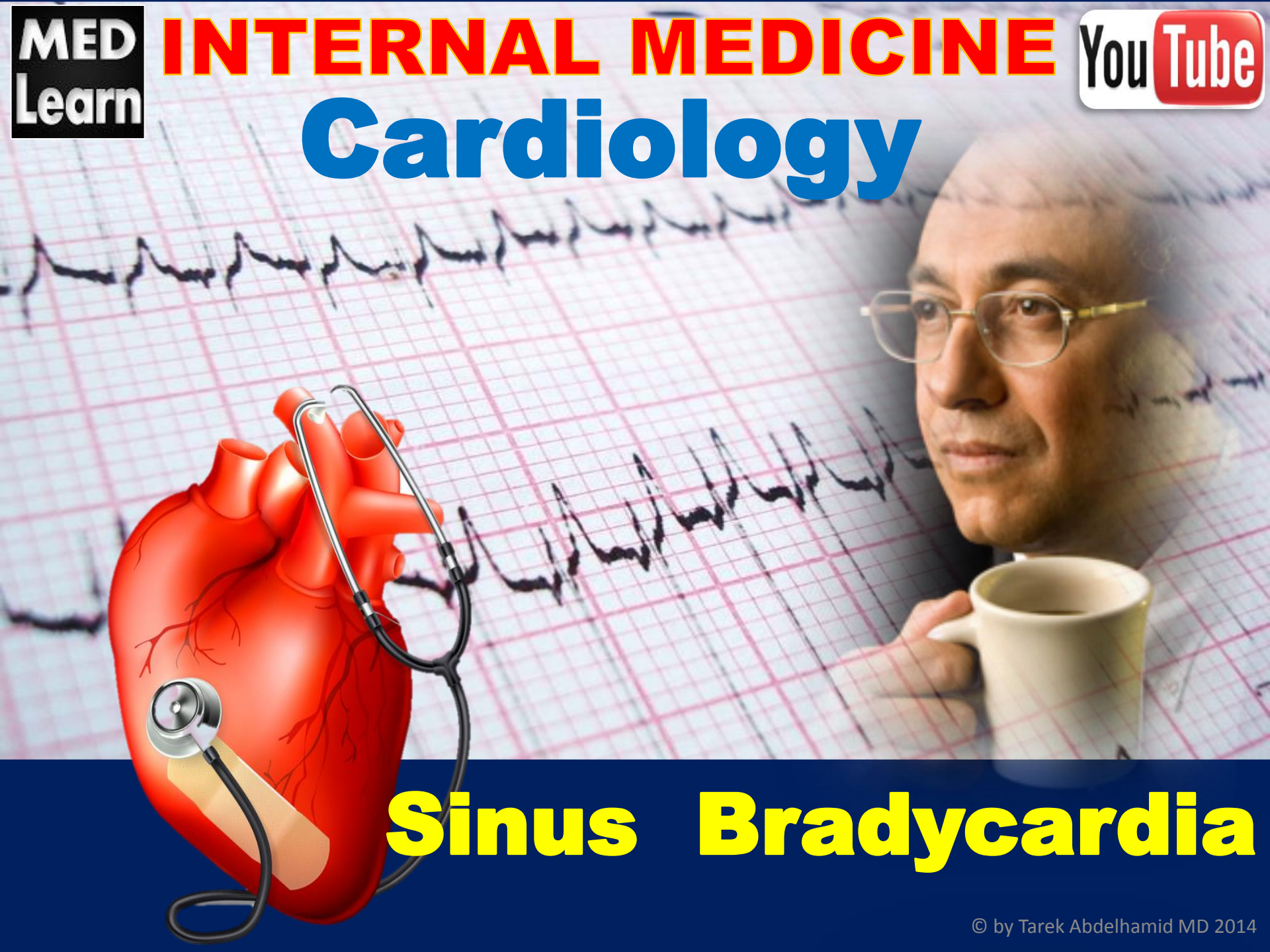
④ Medical treatment to protect the ventricles from the high atrial rhythm include: Digitalis- Beta Blockers- and Ca- Channel antagonists (they can block the AVN to protect the ventricle)

⑤ New innovative therapy include AVN ablation plus pacemaker



NEVER use DC Shock in cases of Digitalis toxicity as it may precipitate VF!

Cardiology



Sinus Bradycardia

Definition

Slow heart rate due to a decrease in impulse formation from the SAN

Aetiology

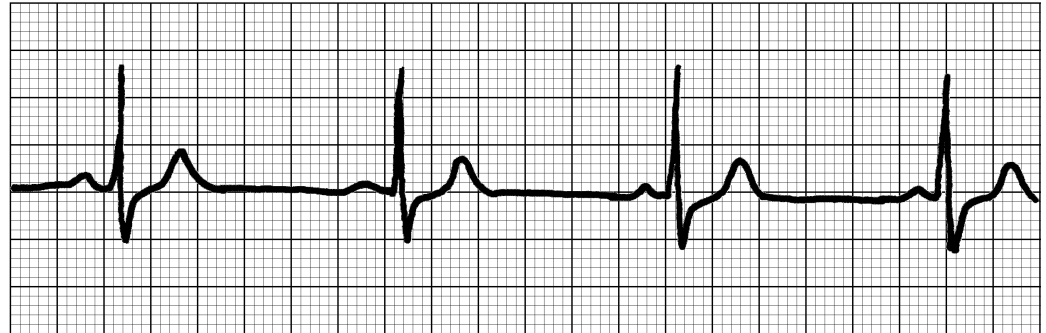
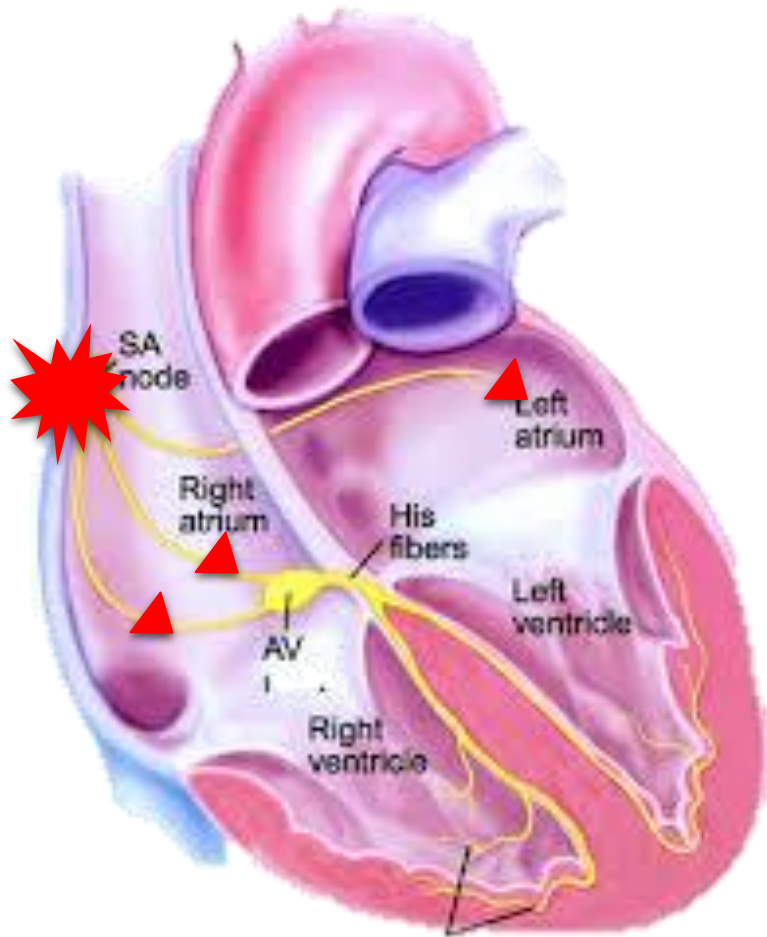
- ① Sick Sinus Syndrome SSS
- ② Increase in Vagal tone (Athletes- Vasovagal attack-Inferior Wall AMI)
- ③ Increase ICP
- ④ Increase Bile Salts e.g. Obstructive Jaundice
- ⑤ Hypothyroidism
- ⑥ Hypothermia

Clinical Picture

Manifestations of the Underlying cause are usually prominent

Special Investigations

Sinus Bradycardia QRS has normal shape

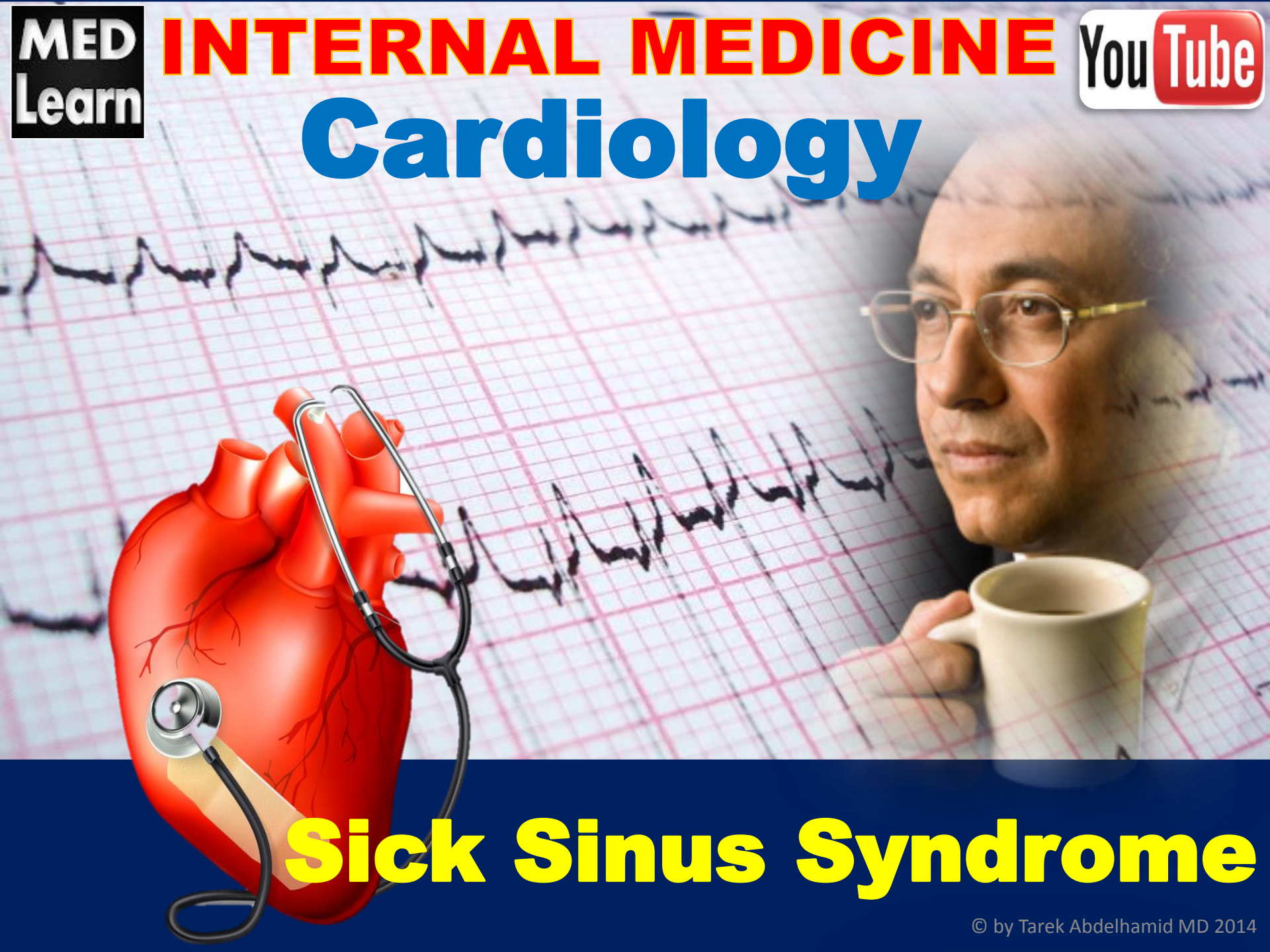


(50 beats/minute)

Treatment

Mainly treatment of the underlying cause

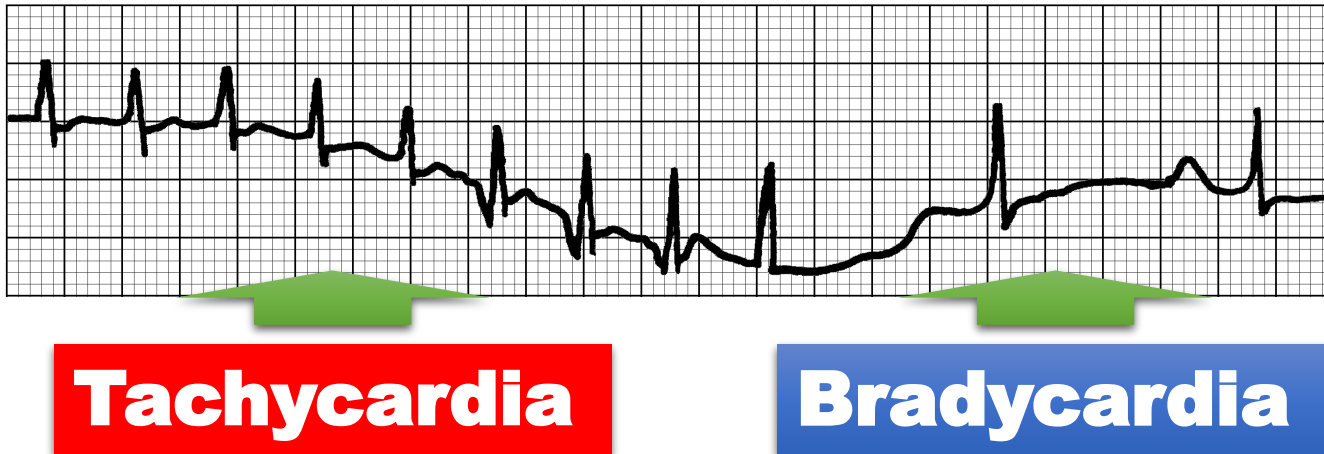
Cardiology



Sick Sinus Syndrome

Definition

A condition affecting the SAN that may cause alternating SB and ST and can make the SA stop working at any moment causing cardiac arrest.

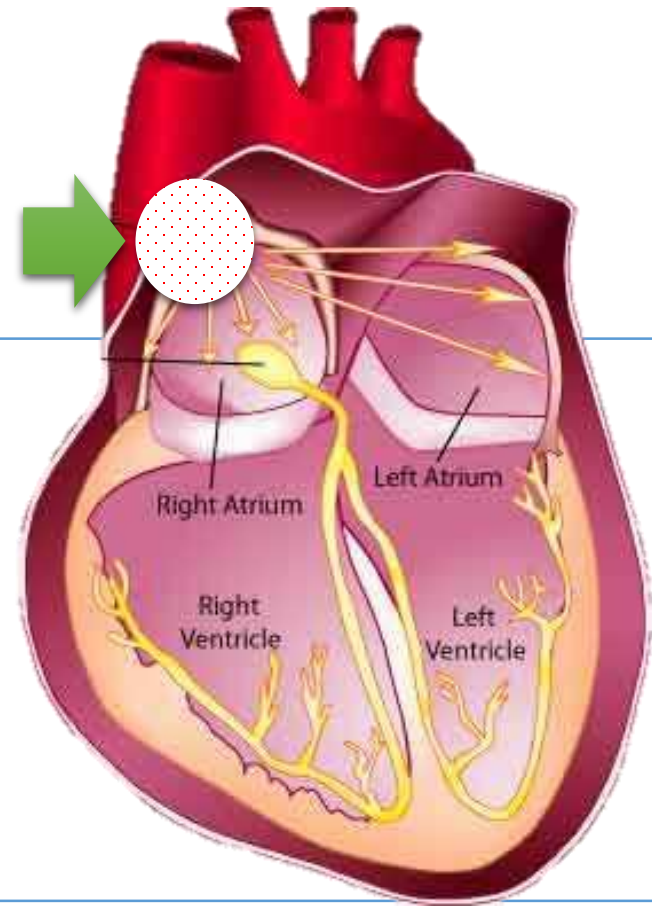


Aetiology

1 Most likely caused by Idiopathic Degeneration and Fibrosis of the SAN

2 Other causes include:

- Amyloidosis
- Sarcoidosis
- Cardiomyopathies
- Chagas disease
- Rarely: Coronary Heart Disease



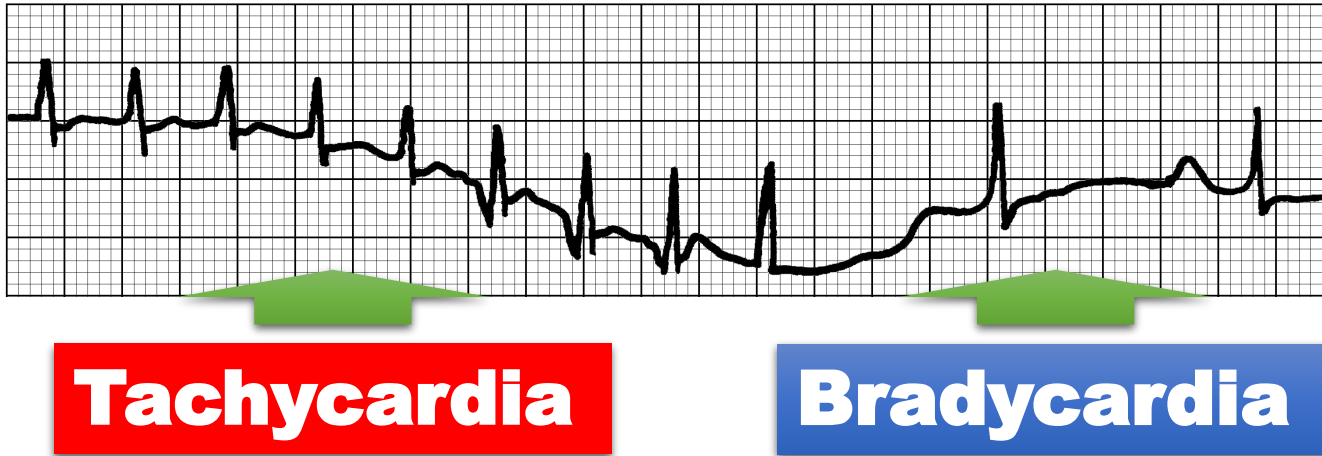
Attacks



Syncope
Dizziness
Confusion
Palpitations

Special Investigations

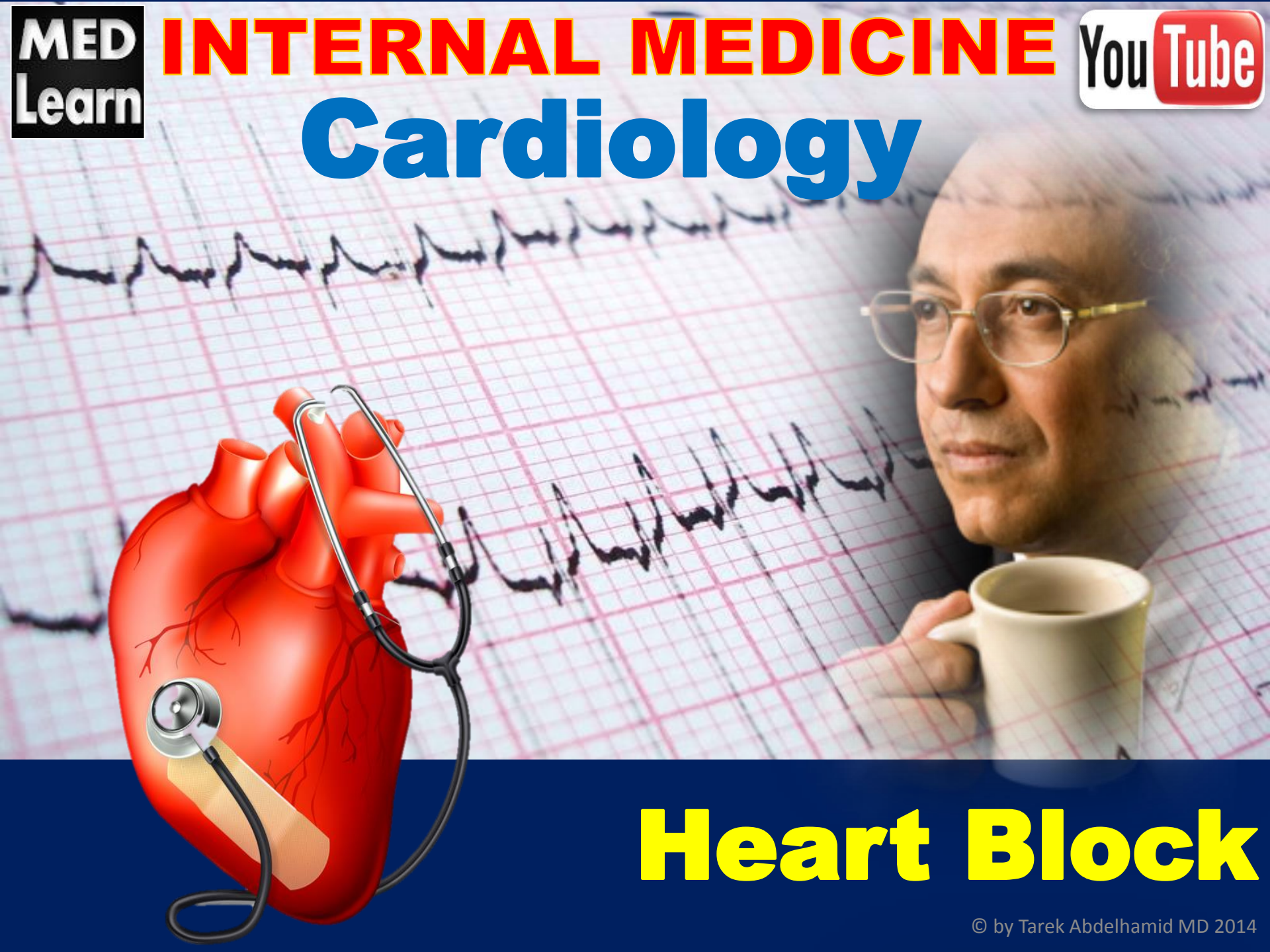
Prolonged ambulatory monitoring of cardiac rhythm is needed to detect the periods of SAN Dysfunction (Holter Monitor)



Treatment

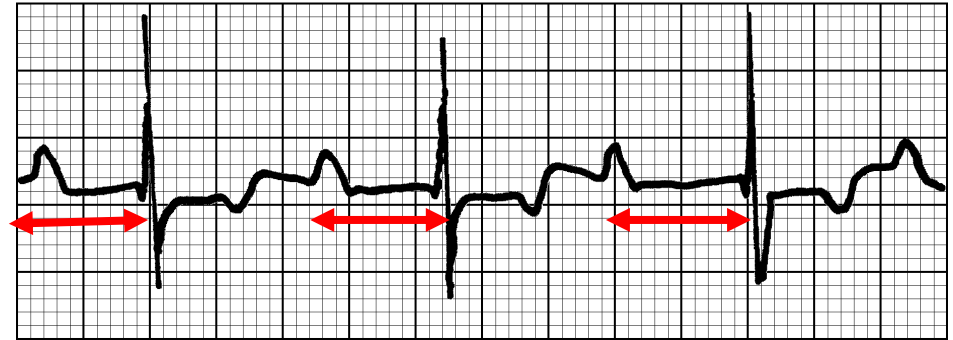
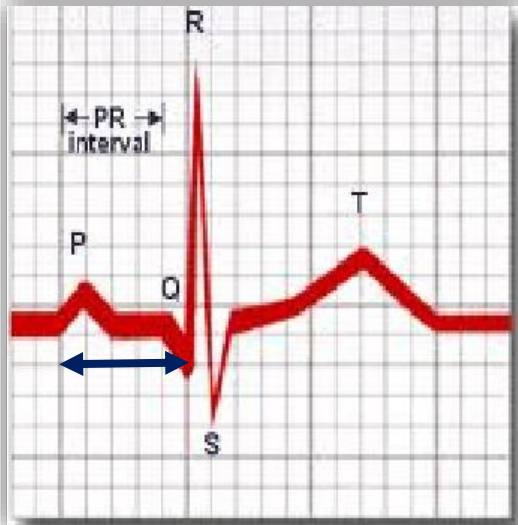
- ① **Permanent Pacemaker** is needed in Most Cases
- ② **Oral theophylline** may be effective especially if Bradycardia is the major manifestation
- ③ **AVOID** drugs that may increase the bradycardia as they may aggravate the condition e.g.
 - Amiodarone
 - Beta Blockers
 - Ca-Channel Blockers
 - Digitalis

Cardiology



Heart Block

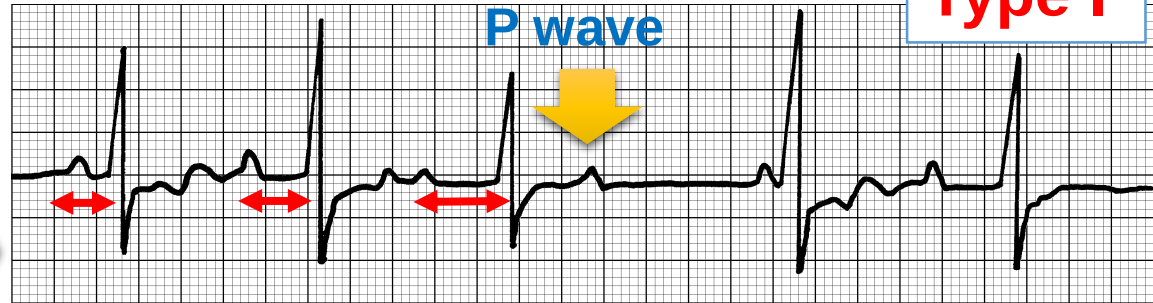
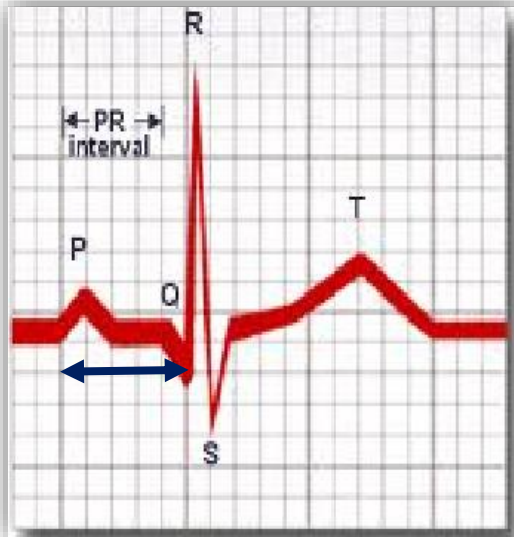
First degree heart block



Prolonged PR intervals in first degree heart block

- ① Associated with prolonged stable PR intervals
- ② May occur in Normal individuals- 2ry to Drugs- or may be seen transiently with organic heart disease
- ③ Usually does not need any specific treatment

Second degree heart block



Type I

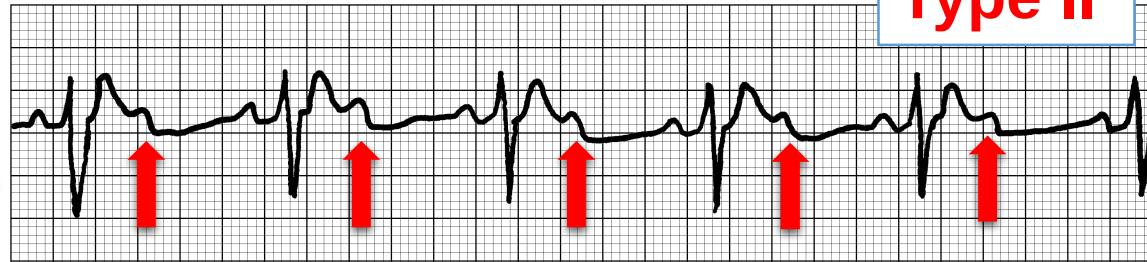
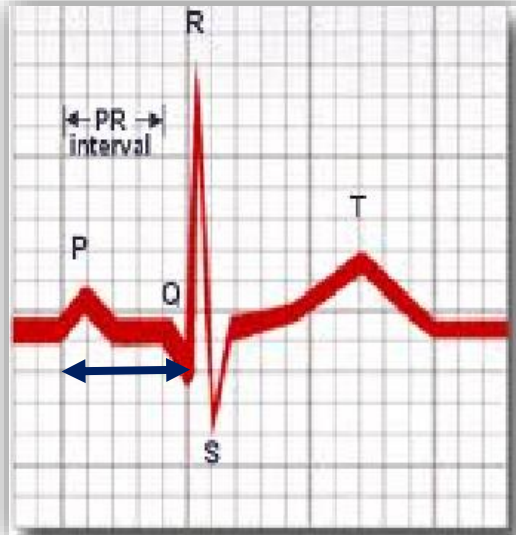
Gradual prolongation of PR intervals followed by non conducted P wave (Dropped beat) in Wenckbach phenomenon

Type I (Mobitz type I block or Wenckbach phenomenon)

- ① Gradual prolongation of PR intervals followed by non conducted P wave (Dropped beat)
- ② Usually not pathological
- ③ Good prognosis
- ④ Usually no treatment is needed....?!

Atropine

Second degree heart block



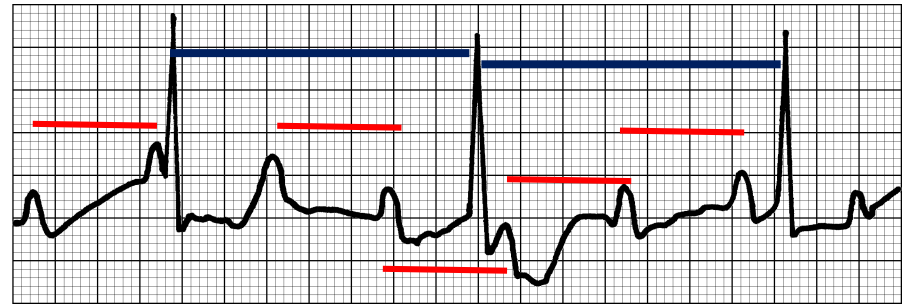
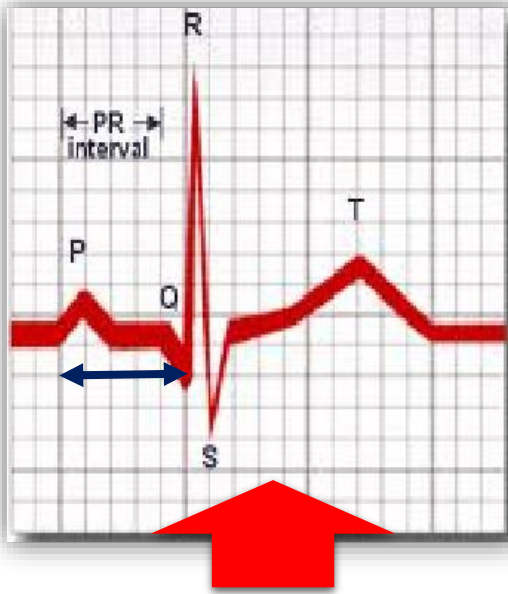
**Occasional non-conducted P waves in
Type II (Mobitz type II block)**

Type II (Mobitz type II block)
Occasional non-conducted P waves



- ① Almost always pathological
- ② Occurs in AMI
- ③ May herald the onset of CHB
- ④ Prophylactic pacing is necessary

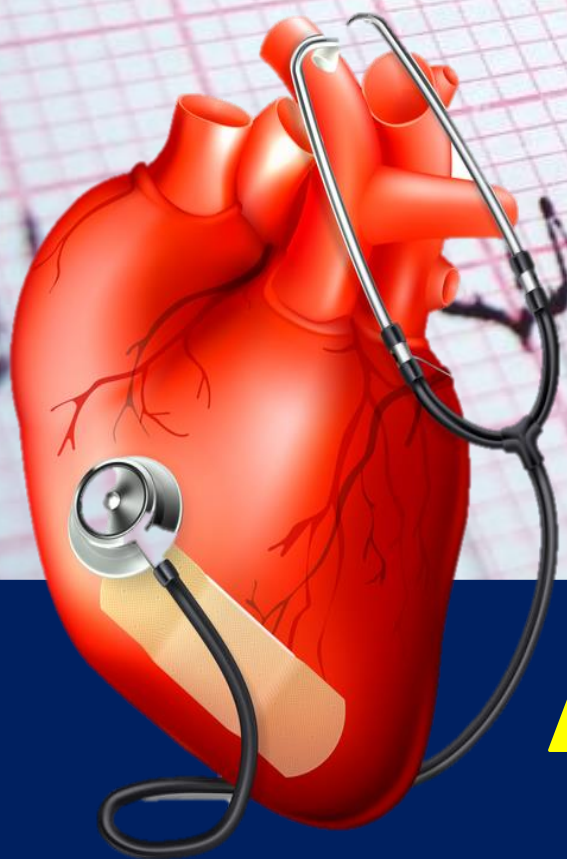
Complete Heart Block **CHB**



P-P intervals are different from R-R intervals.

- ① Complete atrio-ventricular dissociation as the ventricle follows IVR while atrial activity follows an atrial focus.
- ② Causes include: **AMI**- Gumma of the Septum in Syphilis- and Calcification (e.g. Aortic Stenosis)
- ③ Bradycardia
- ④ Permanent Pacemaker is the treatment of Choice!

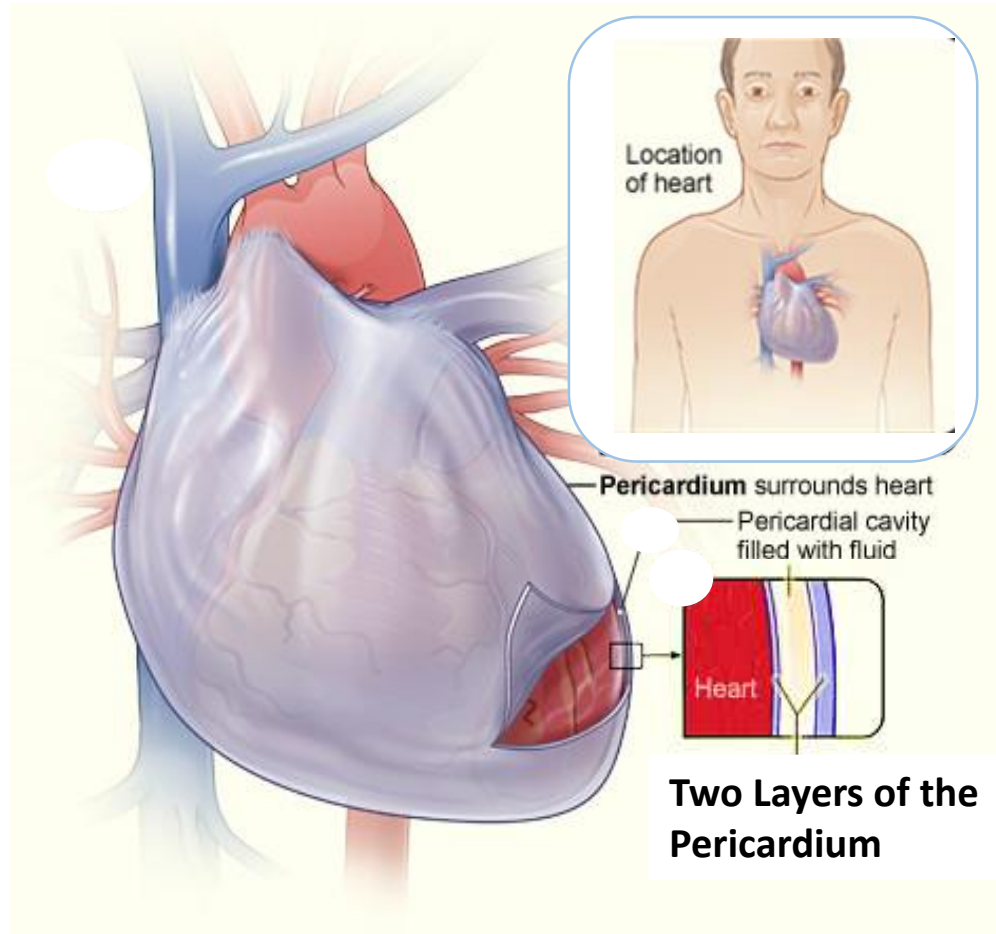
Cardiology



Acute Pericarditis

Definition

Acute inflammatory condition affecting the Pericardium

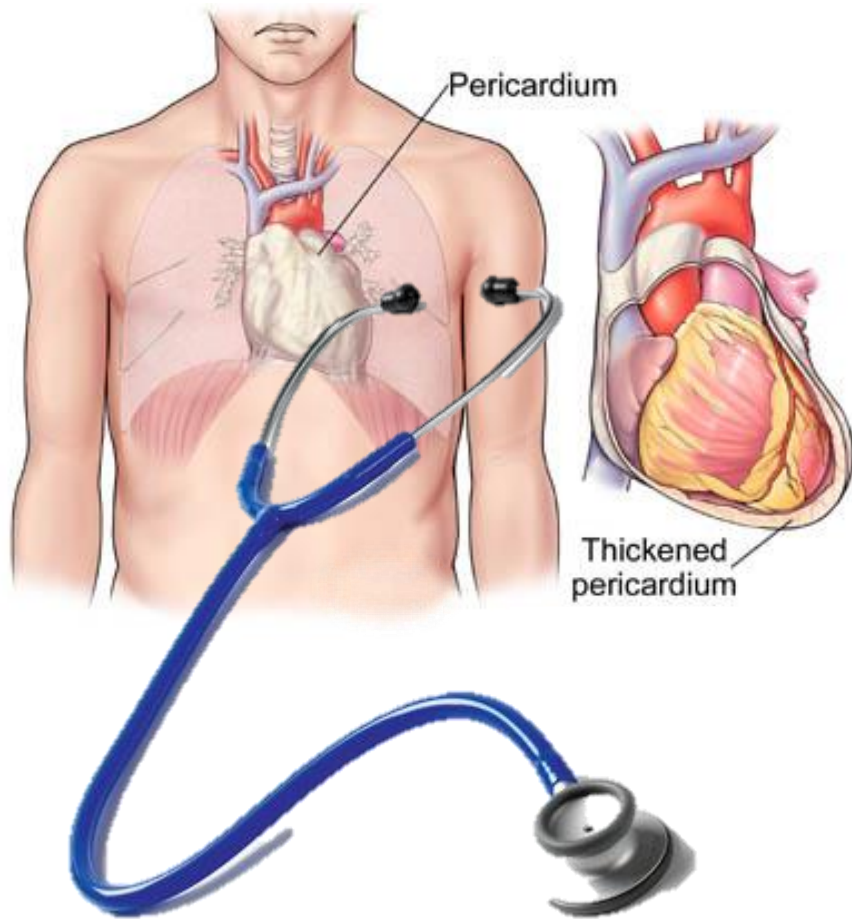


Aetiology

The condition could be caused by:

- ① Infections (**Viral**- Fungal- and TB)
- ② Immune Process e.g. Rheumatic Fever- Collagen Diseases (SLE)- Post-MI (Dressler Syndrome)
- ③ Others such as Uremia- Leukemic Infiltration- Drugs (e.g. Hydralazine and Procainamide)

Clinical Picture



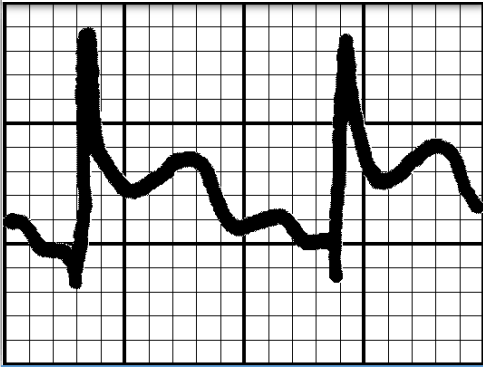
Pain

- ① Retrosternal
- ② Continuous
- ③ Stitching or dull ache
- ④ Radiating to the left shoulder
- ⑤ Increases by Inspiration and swallowing
- ⑥ Associated with fever and leukocytosis

**Pericardial friction Rub
could be heard on
Auscultation**

Special Investigations

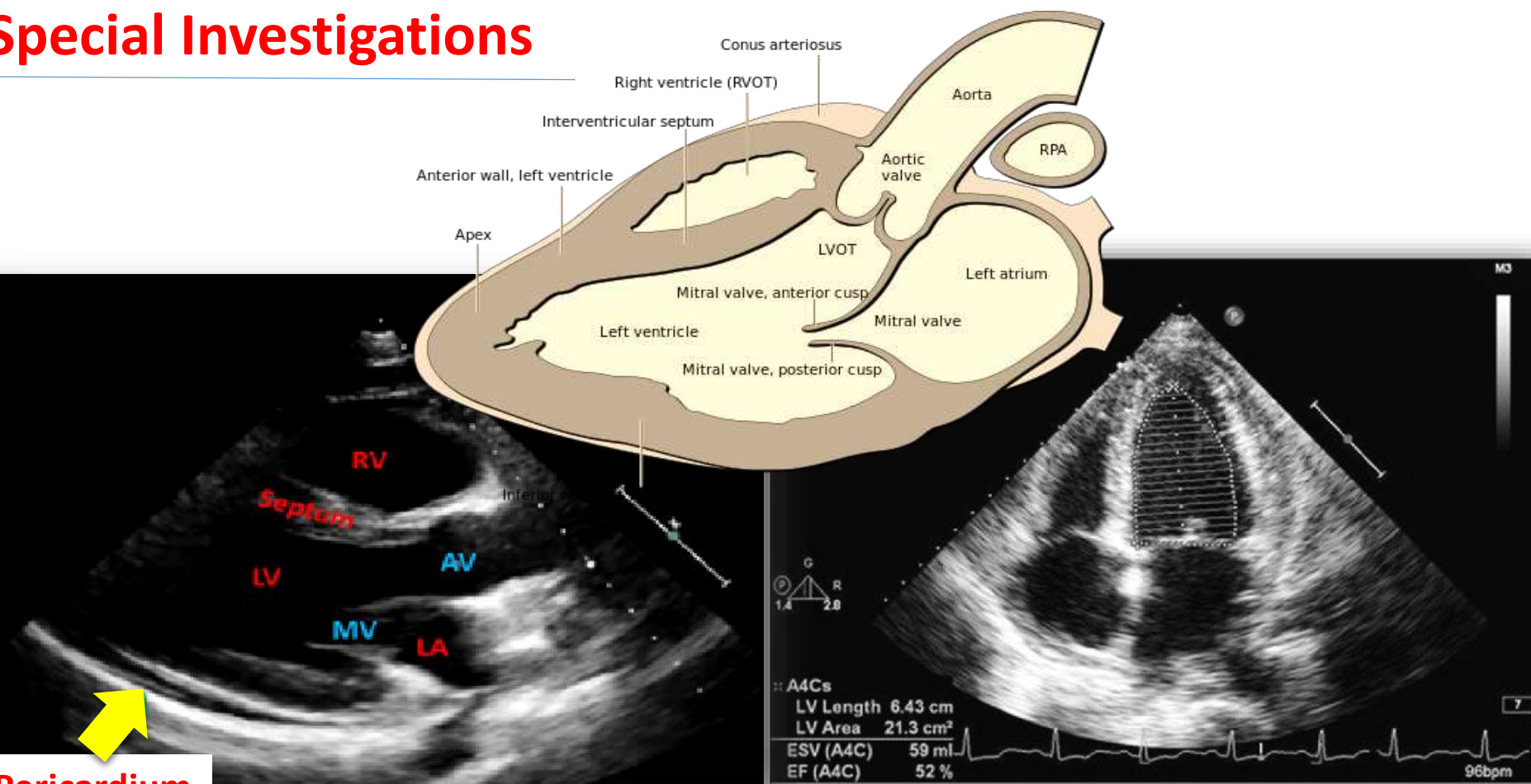
**ST segment
Elevated**



**Concave
Upward**



Special Investigations



Echo Can detect the presence of associated small pericardial effusion

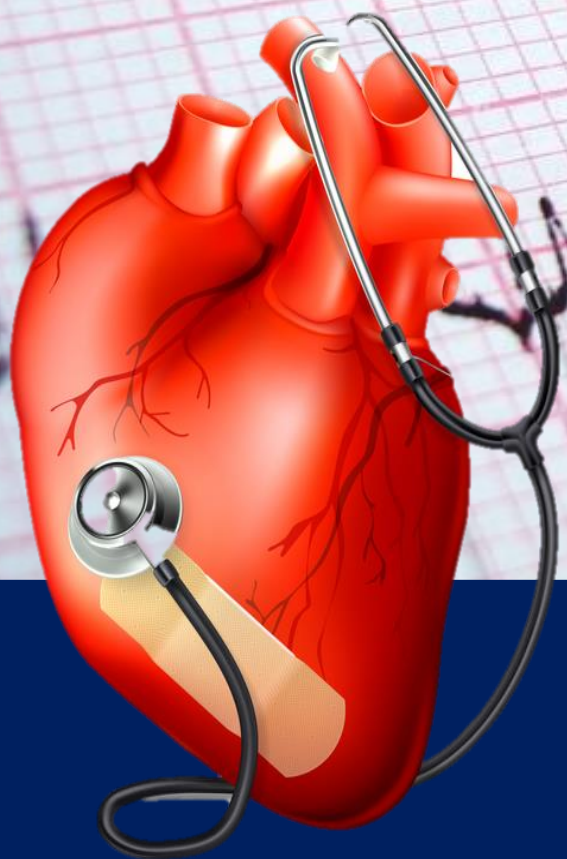
Treatment

- **Treatment is mainly by:** Aspirin- NSAID- OR Glucocorticoids to decrease the pain
- **Colchicine** to prevent recurrence
- Correcting the cause is essential



AVOID Anticoagulants as they may cause bleeding in the inflamed pericardium causing fatal cardiac tamponade

Cardiology

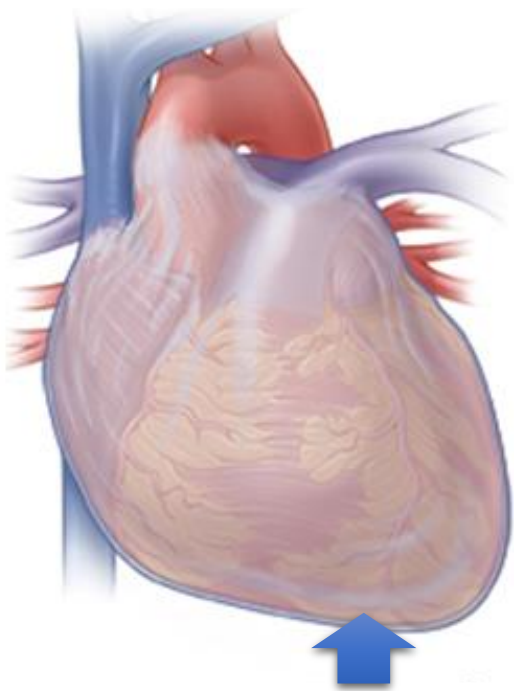


Pericardial Effusion

Definition

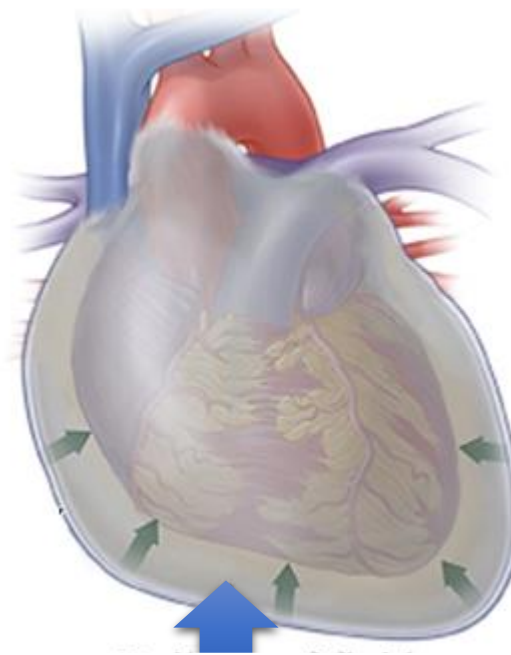
Accumulation of fluid inside the pericardial sac

Normal Heart



Pericardium

Pericardial Effusion



Accumulation of fluid

Aetiology

This condition can be caused by nearly all causes of acute pericarditis e.g. Viral- TB- etc. (See acute pericarditis)

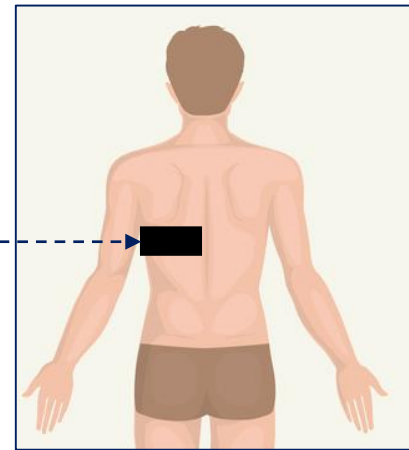
Clinical Picture

**Congested
Neck veins
with slow Y
Descent**

**Chest pain
(Dull aching)**

Ewart's sign

Area of Lung
collapse beneath
the angle of left
scapula



**Distant heart
sounds and weak
or absent Apex
beat**

SCS

**Oedema of
Lower
Limbs**

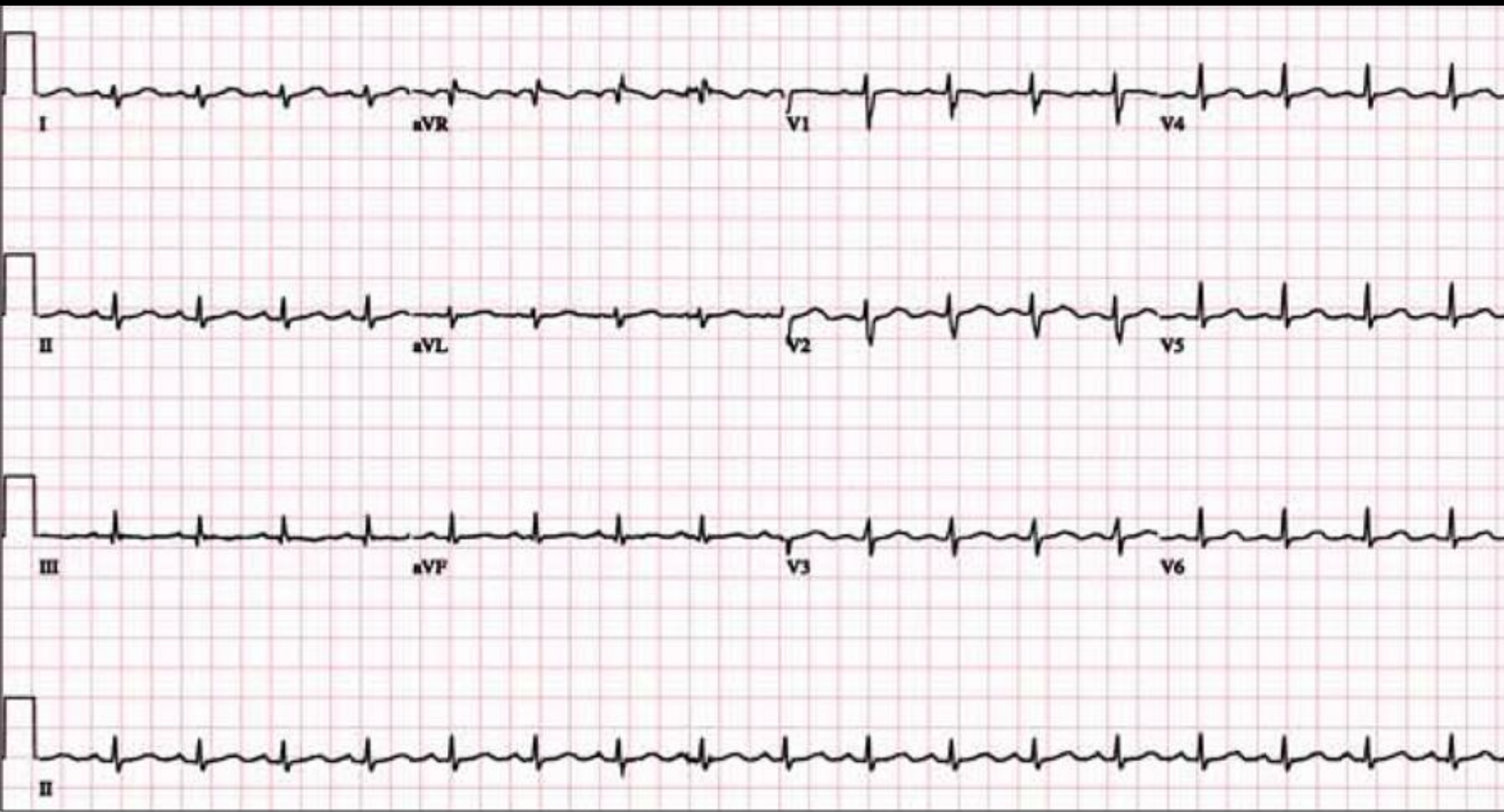
LCO
Weak
pulse

Pulsus Paradox
Decrease pulse volume
during inspiration



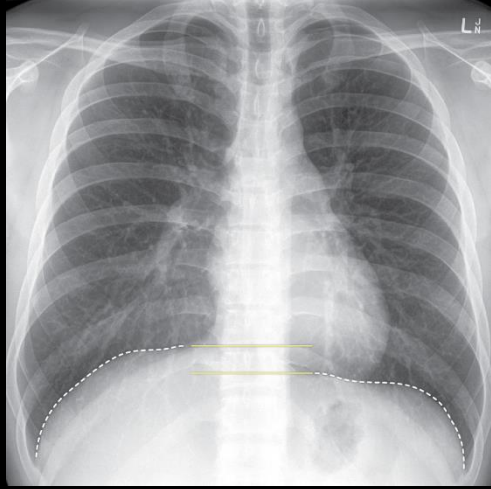
Special Investigations (1)

Low voltage ECG in Pericardial Effusion



Special Investigations (2)

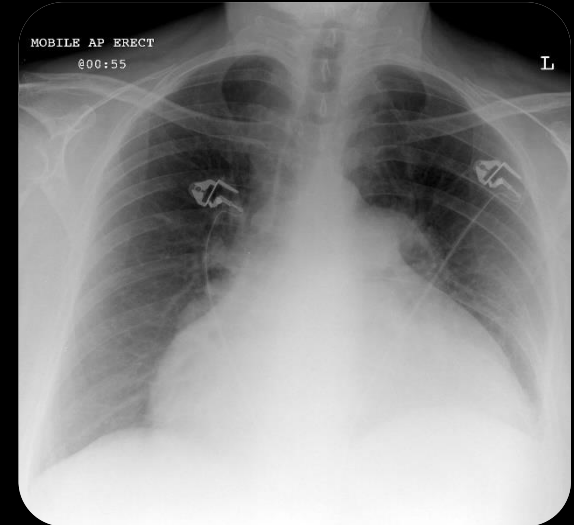
X-Ray



Normal



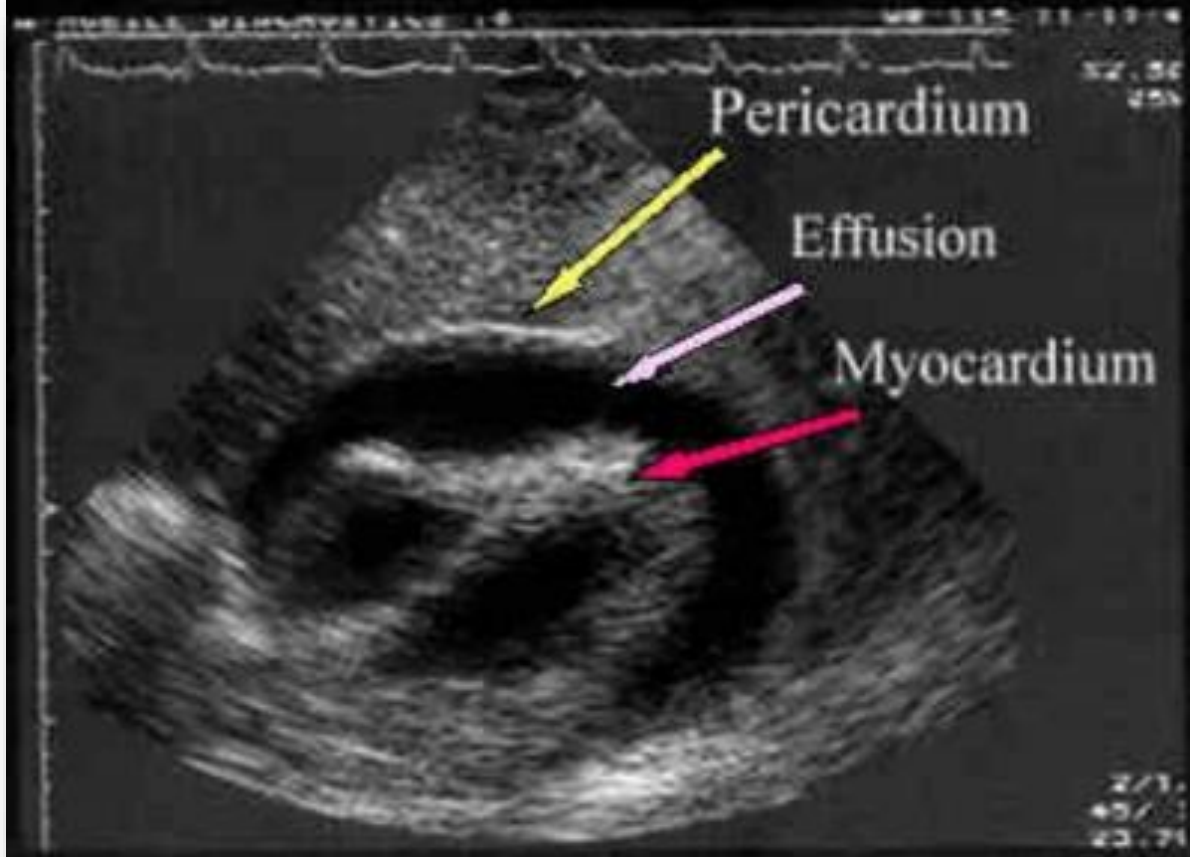
LVH



Pericardial Effusion

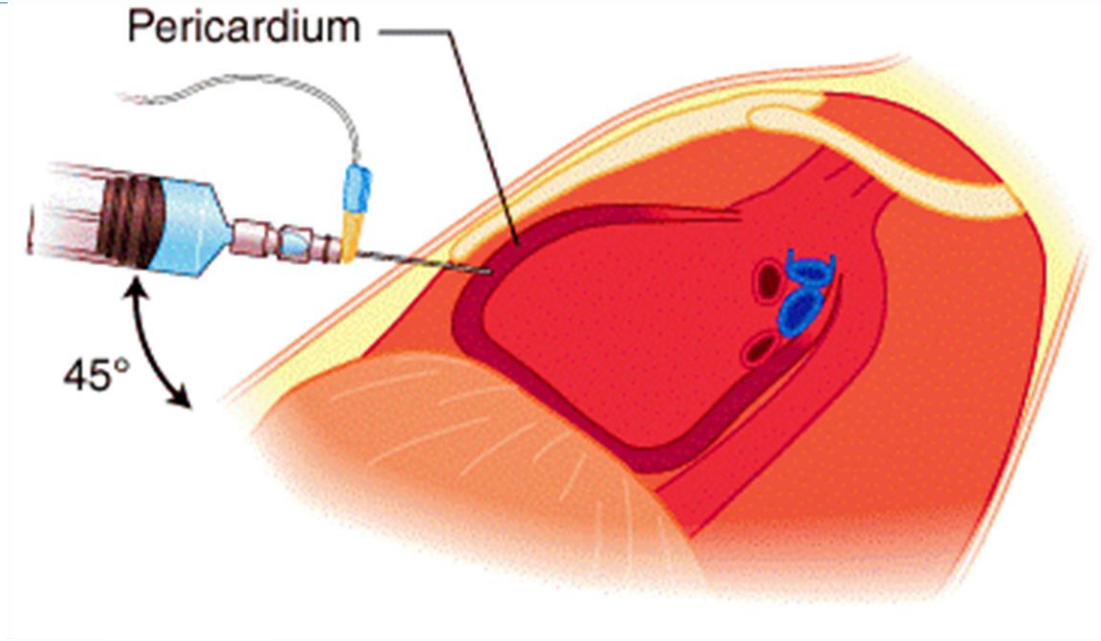
Special Investigations (3)

Echocardiography

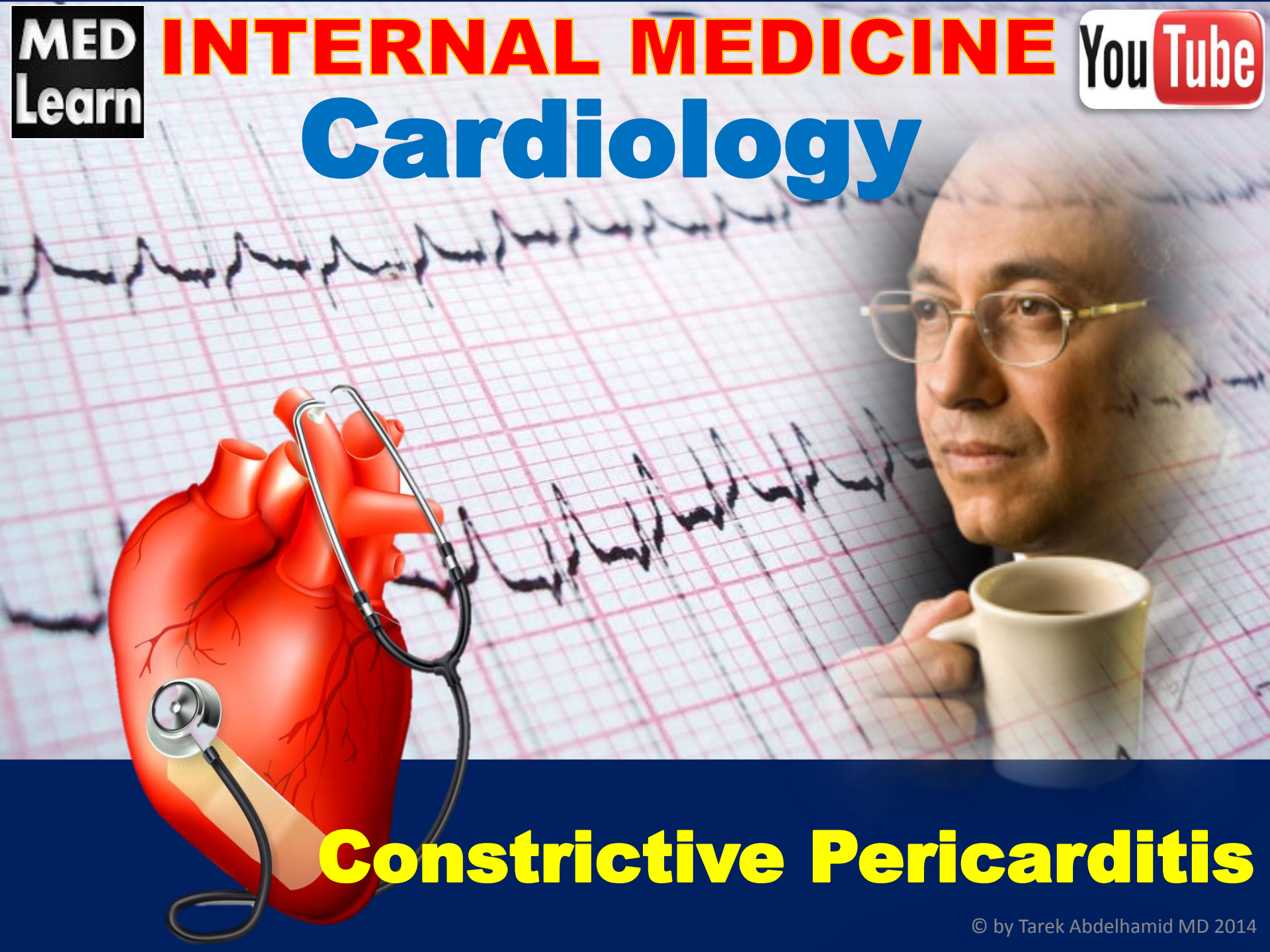


Treatment

Aspiration (Diagnostic and therapeutic) *plus* treatment of the cause (essential)



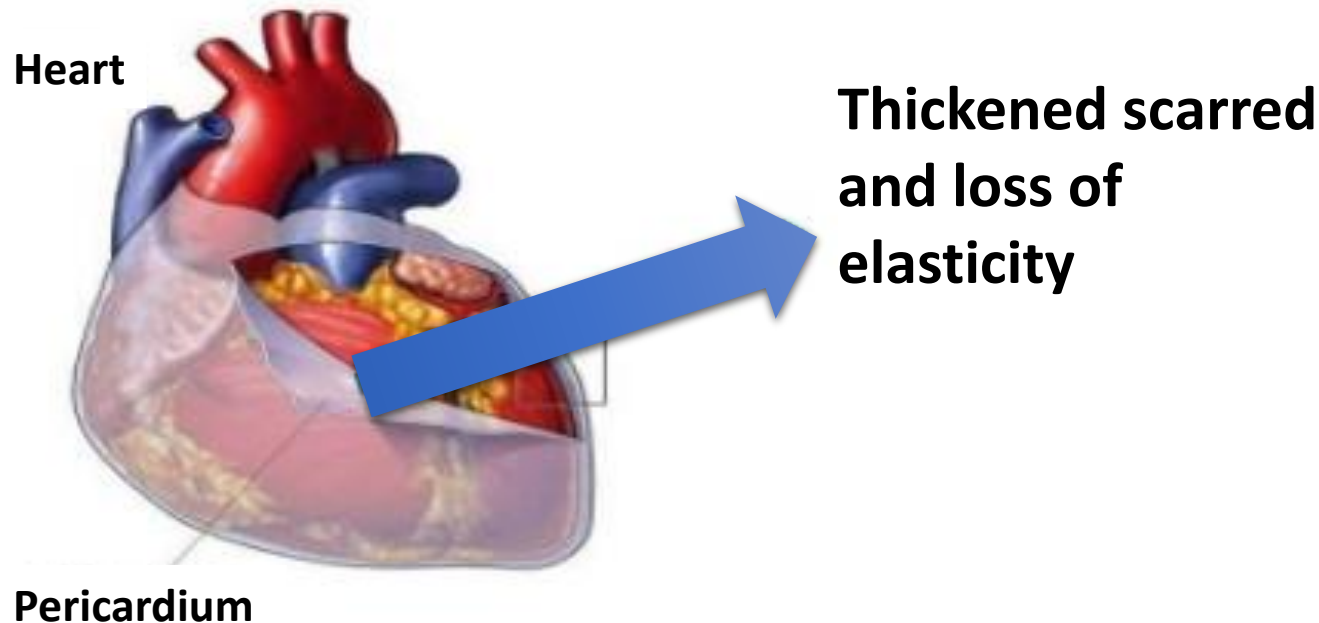
Cardiology



Constrictive Pericarditis

Definition

Constrictive Pericarditis represents a condition in which the pericardium changed into a fibrotic sac that surrounds the heart and prevents it from expanding fully during diastole.

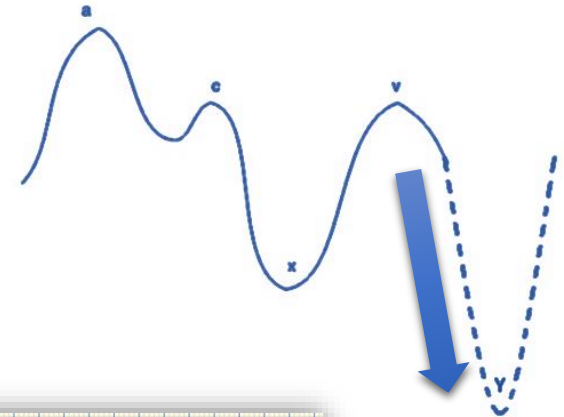


Aetiology

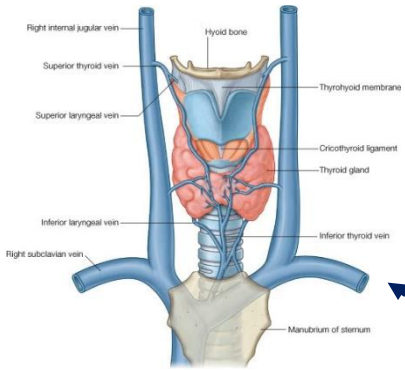
The condition essentially occurs as a sequel to inflammatory process affecting the pericardium e.g. after acute pericarditis (Viral, T.B. , ..., ..., ...)

Clinical Picture

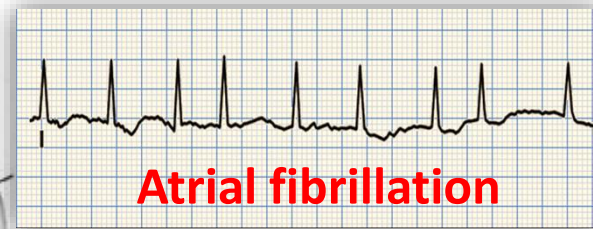
**Congested Neck Veins
with Rapid Y Descent**



**LCO: Headache-Weakness-
Fatigue-low blood pressure**



Pericardial Knock



**Congested
Enlarged
Tender Liver**

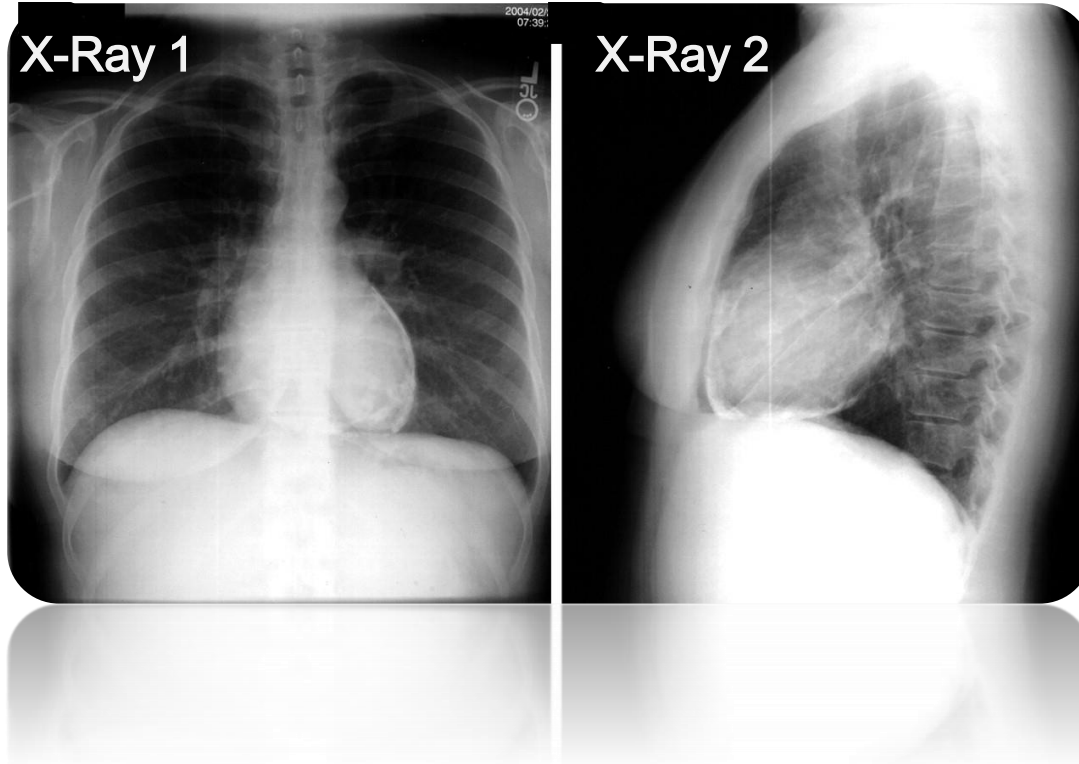
**Abdominal
Pain**

Ascites

**Oedema of
Both LL**

Special Investigations

Radiology: X-Ray, CT & MRI can detect the thickened fibrotic pericardium

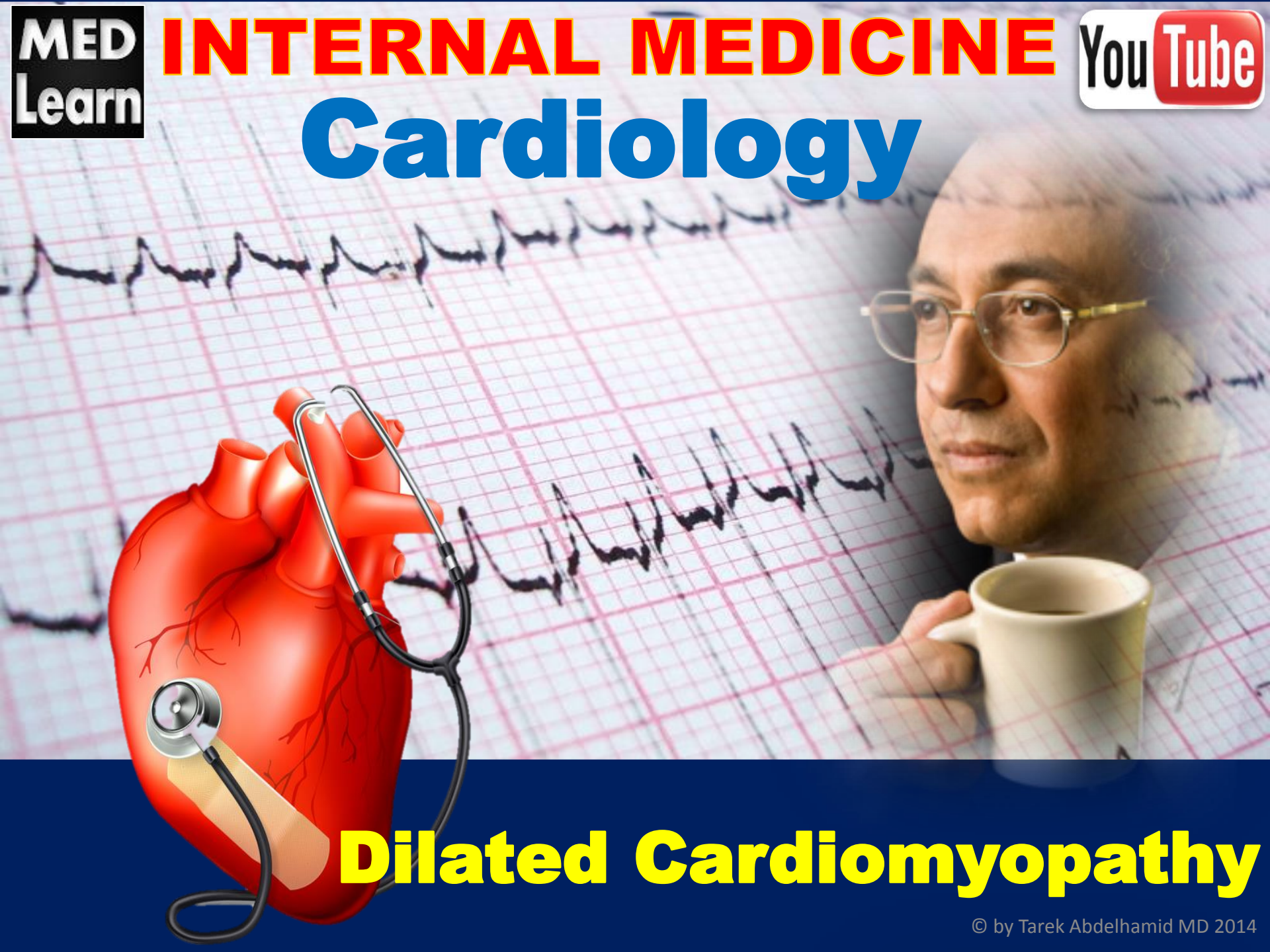


Treatment

Usually, No Medical treatment is needed

**Definitive care is primarily surgical
(i.e. pericardiectomy)**

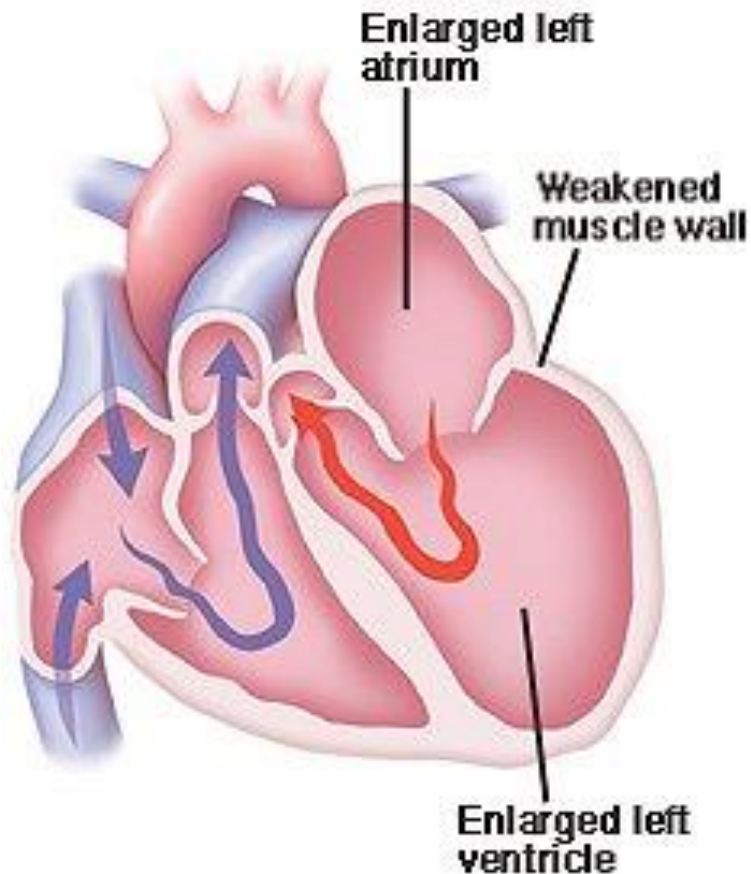
Cardiology



Dilated Cardiomyopathy

Definition

A disease affecting cardiac muscles and is characterized by dilatation of cardiac chambers.

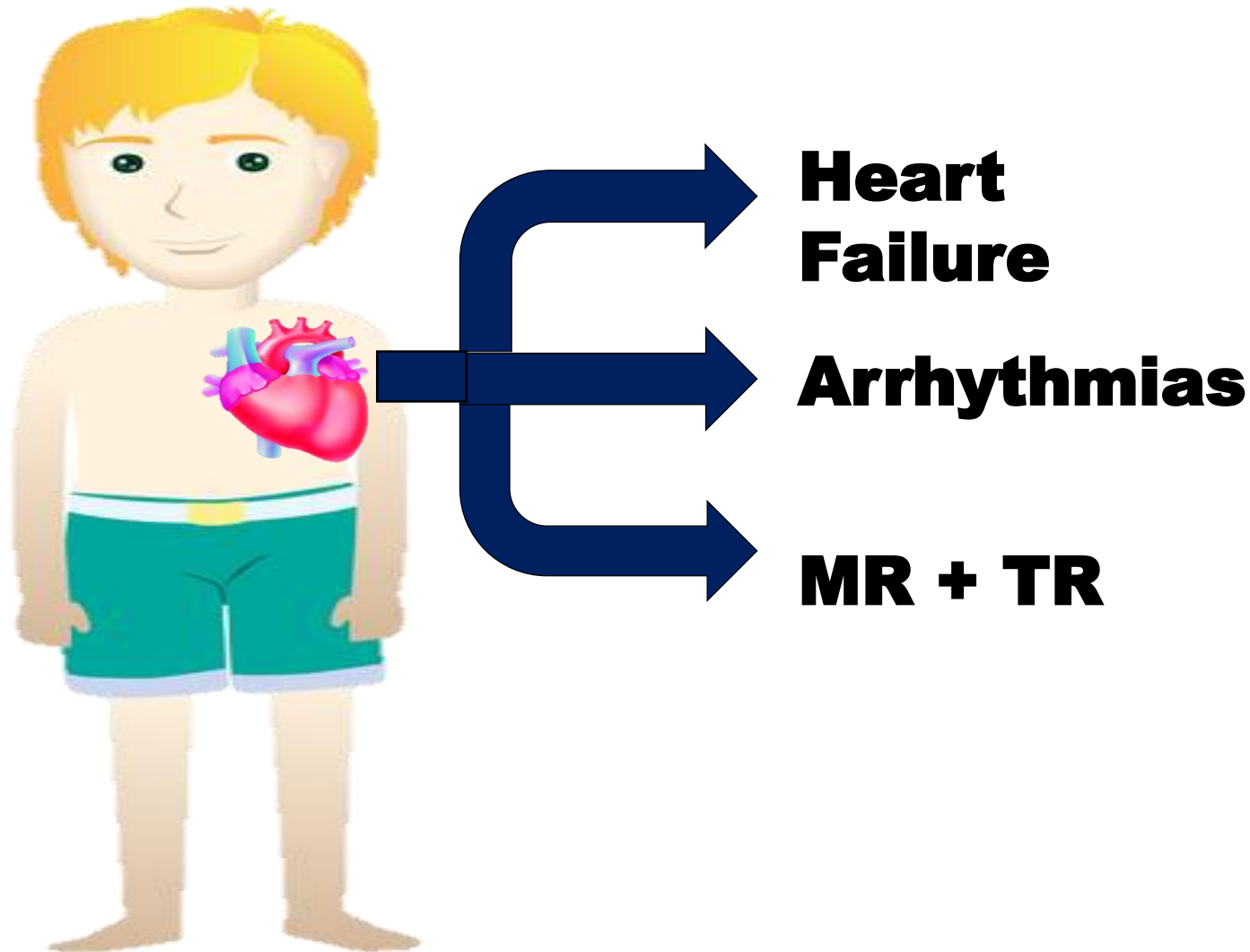


Aetiology

- ① Alcohol
- ② Post-Myocarditis
- ③ Post-Partum (?! Immune)
- ④ Diabetes Mellitus
- ⑤ Drugs: Doxorubicin
- ⑤ Idiopathic
- ⑥ Familial: with Multiple gene mutations that affect encoding of myocardial structural proteins.

Clinical Picture

When Heart Failure develops for unknown cause above the age of 40 YOU should suspect dilated cardiomyopathy



Special Investigations

- 1-Echo: shows LV Dilatation and dysfunction**
- 2-Endomyocardial biopsy (Fat cells)**
- 3-Exclude any accountable factor e.g. thiamine deficiency (Vitamin B1)**

Echo

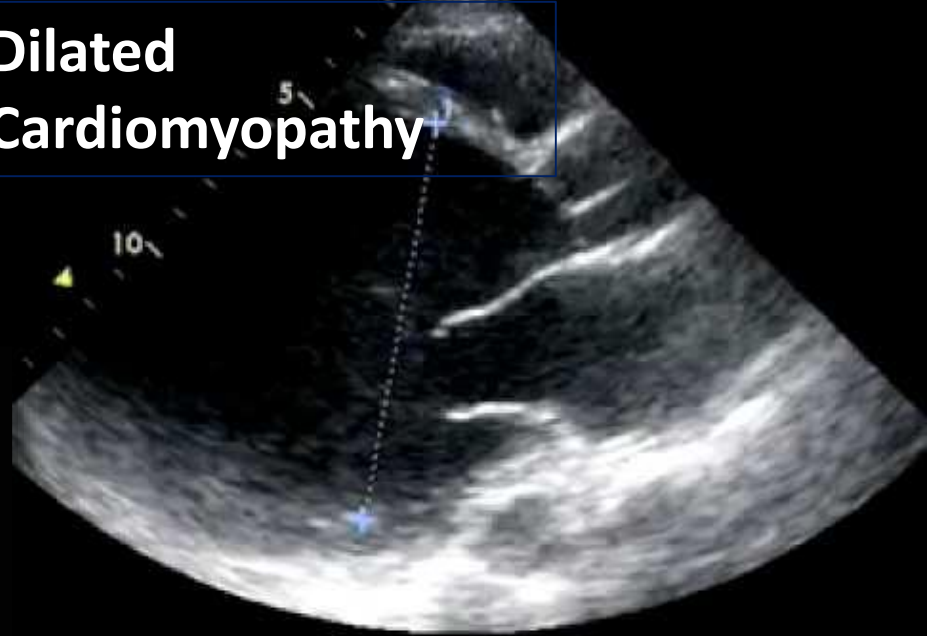
Normal

DIOLOGY 301730
[E"
.D

RV
LV
AO
LA



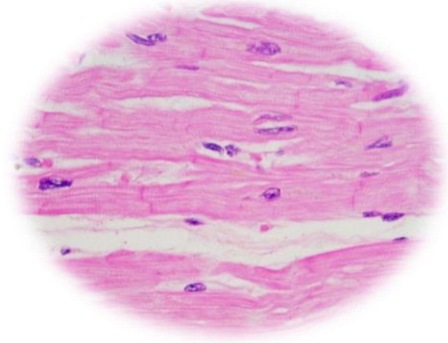
**Dilated
Cardiomyopathy**



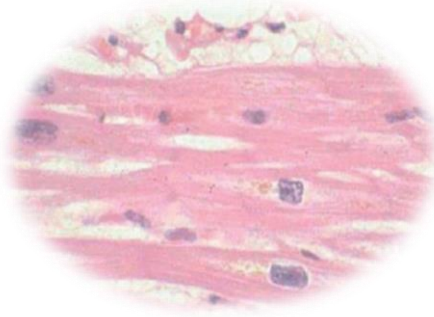
Endomyocardial biopsy



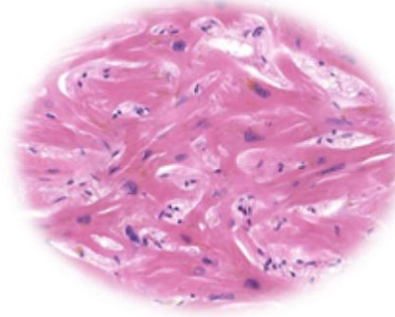
Normal



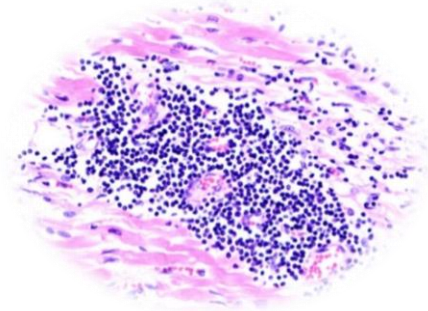
**Dilated
Cardiomyopathy**



**Hypertrophic
Cardiomyopathy**



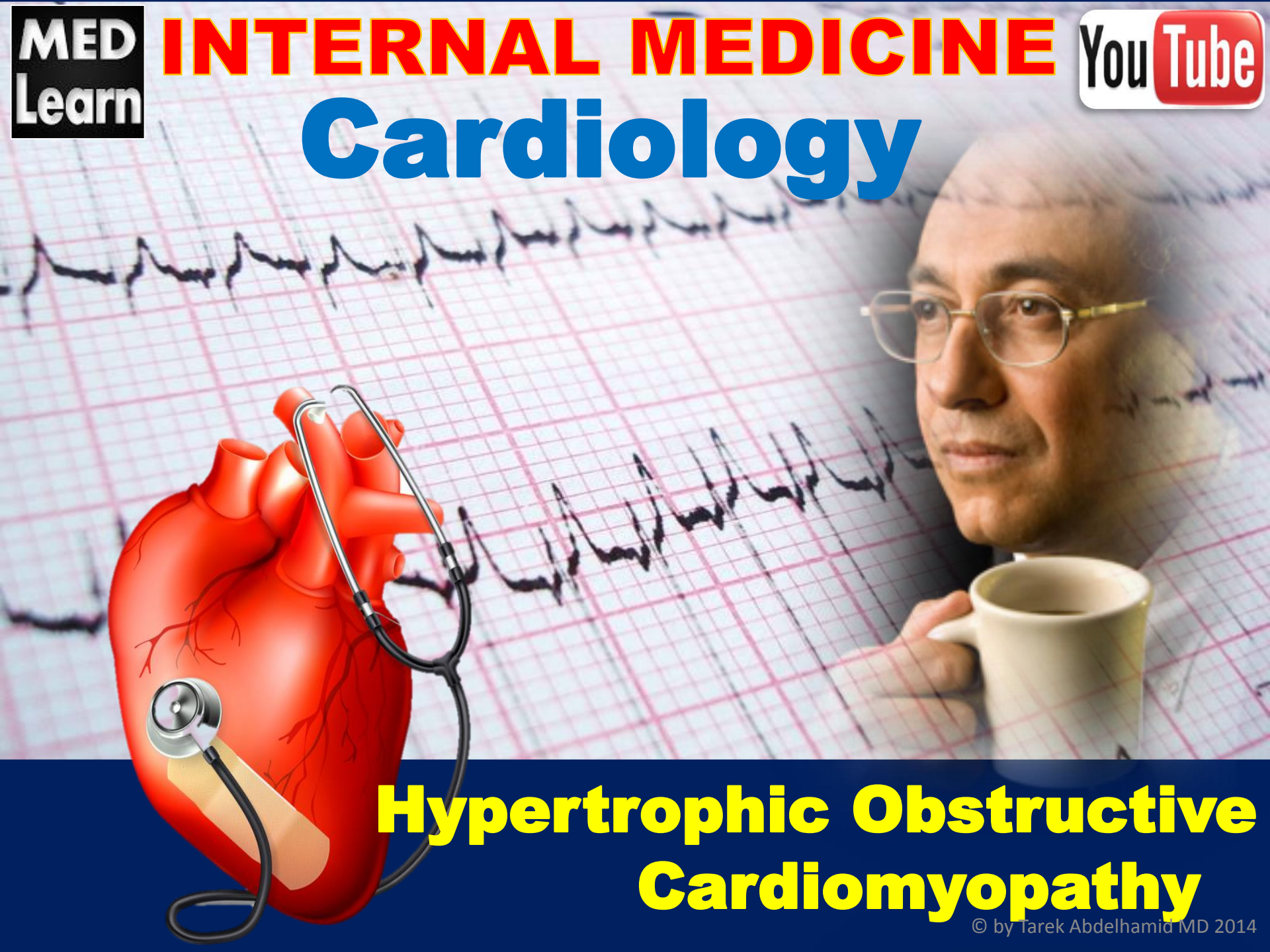
Myocarditis



Treatment

- ① No specific treatment is available
- ② Treatment of associated arrhythmias
- ③ **STOP** Alcohol (Some elements of reversibility in cases of alcoholic cardiomyopathy)

Cardiology

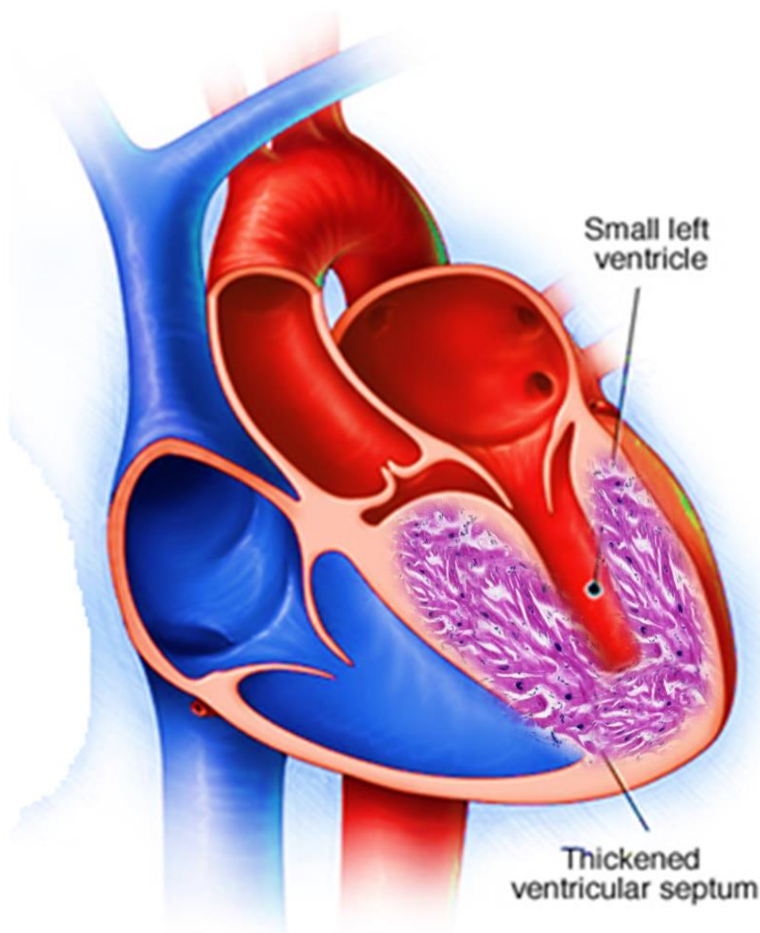


Hypertrophic Obstructive Cardiomyopathy

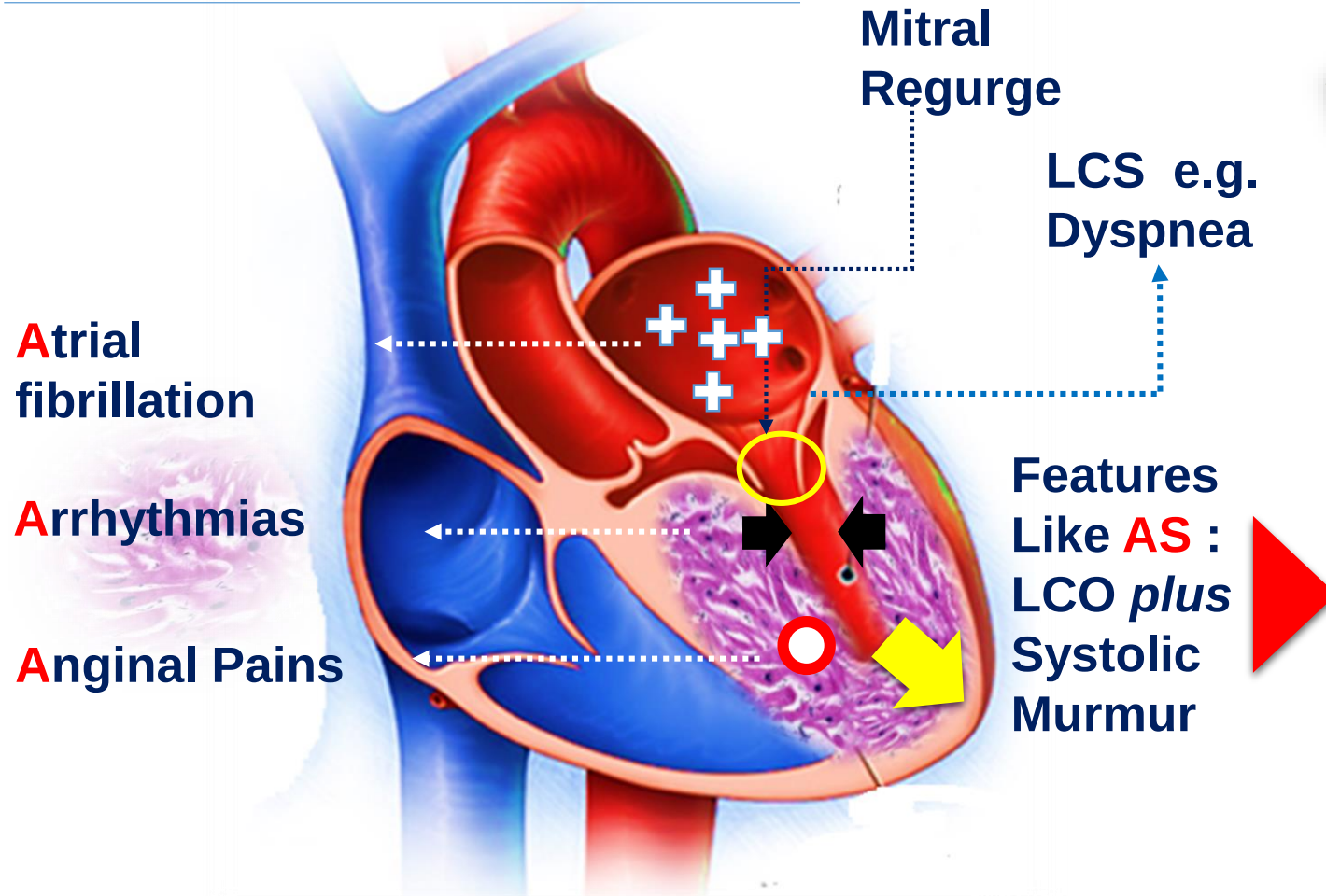
Definition

HOCM is a myocardial disorder characterized by:

- ① Ventricular Hypertrophy,
- ② Diminished LV cavity, and
- ③ Impaired relaxation 2ry to disarray of ventricular muscle fibers.



Clinical Picture



**Syncope
with
exercise**



Avoid Digitalis
Avoid Nitrates
Avoid Strenuous exercise

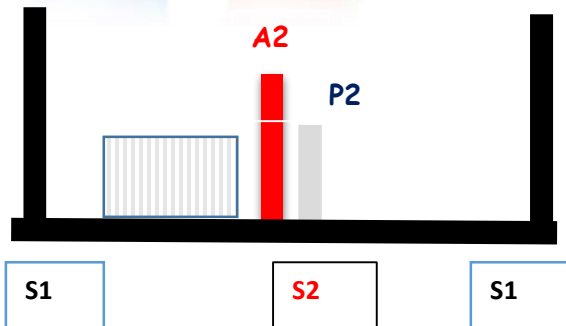
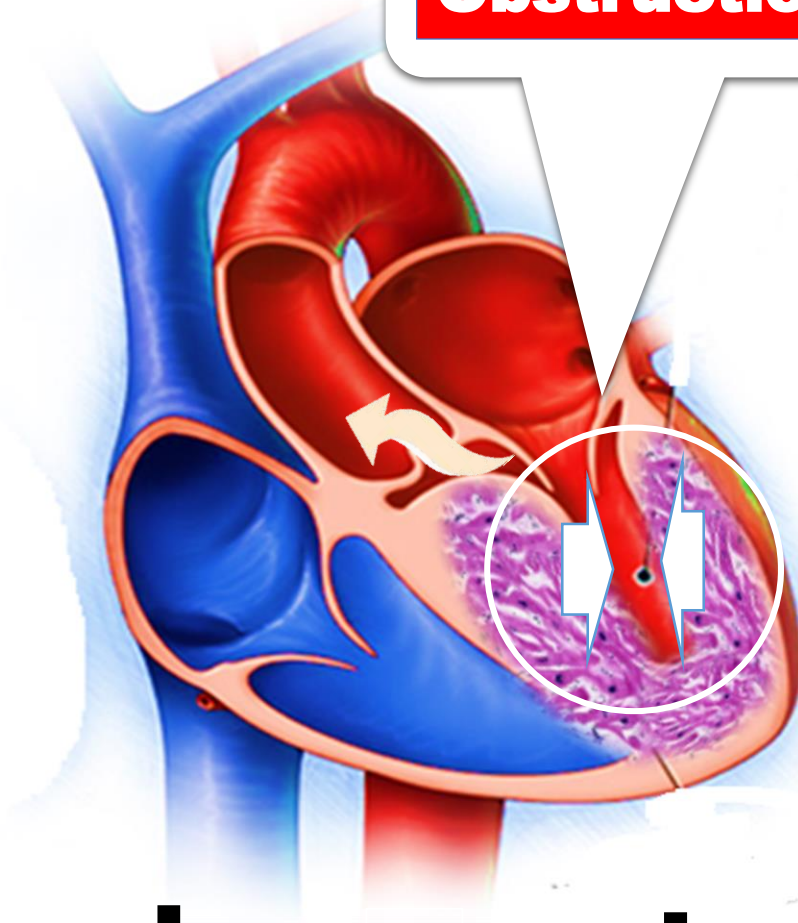
Dynamic Outflow Obstruction



- ① Increase myocardial contractility (**Strenuous exercise**)
- ② Decrease size of the heart e.g. Decrease VR such as (**Standing-Nitrates- Valsalva Maneuver**)
- ③ Digitalis

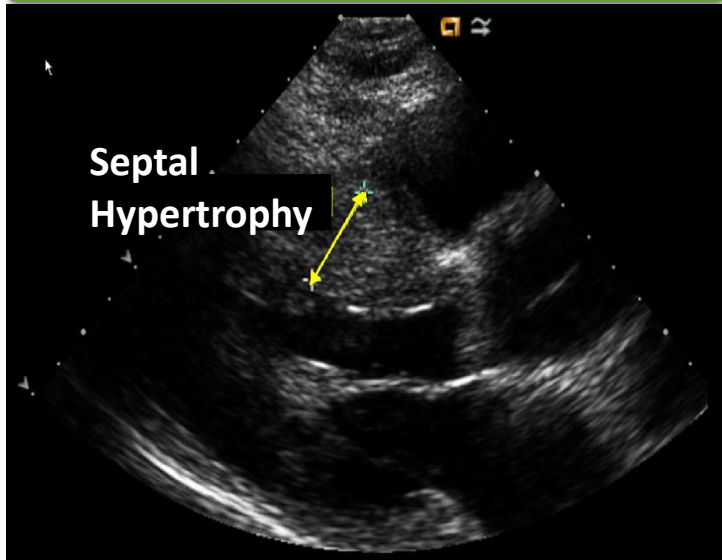


Beta Blockers- Verapamil

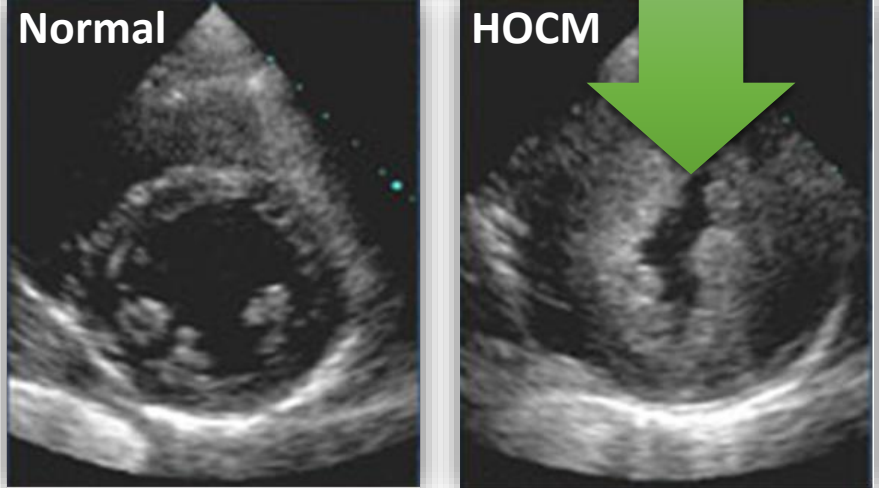


Special Investigations

Echo: Shows Asymmetrical Septal Hypertrophy



Diminished LV Cavity



Treatment

① **Beta Blockers**: should be used initially for symptomatic cases

② **Verapamil**:

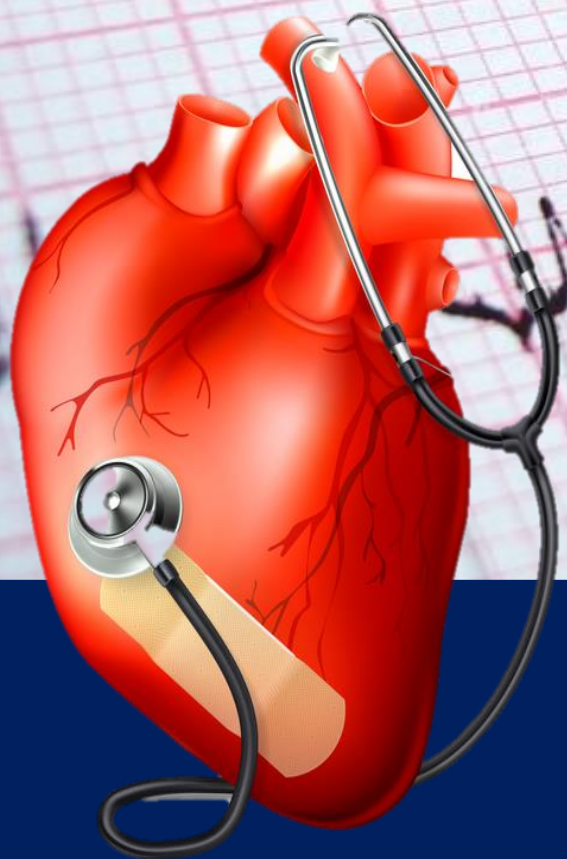


Myocardial contractility
Myocardial Oxygen needs
Arrhythmias

③ **Anticoagulants** (if Af developed)



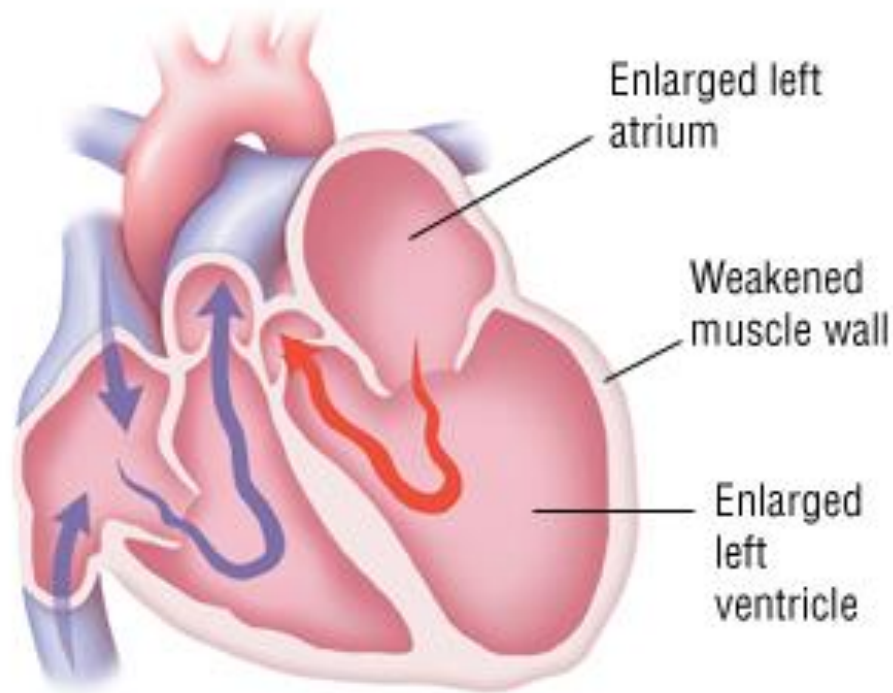
Cardiology



Restrictive Cardiomyopathy

Definition

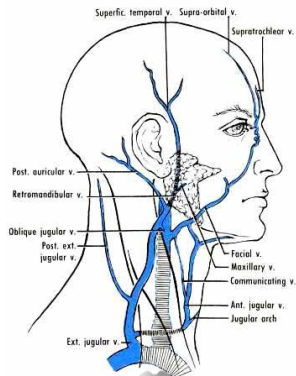
A pathological condition of cardiac muscles that “restricts” cardiac dilatation during diastole thus causes predominantly diastolic cardiac dysfunction with relatively preserved systolic functions.



Aetiology

- ① **Amyloidosis**
- ② **Irradiation**
- ③ **Carcinoid Syndrome**
- ④ **Sarcoidosis**
- ⑤ **Hemochromatosis**
- ⑤ **Scleroderma**

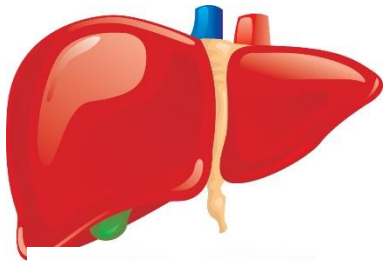
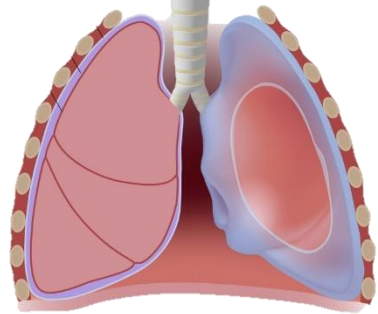
Clinical Picture



**Congested
Neck Veins**

LCS

Inability of the heart to dilate properly or (fill) during diastole causes LCS (Dyspnea) or SCS (Dependent Oedema- Congested Hepatomegaly- Congested Neck Veins)



**Congested
Hepatomegaly**



**Dependent
Oedema-**

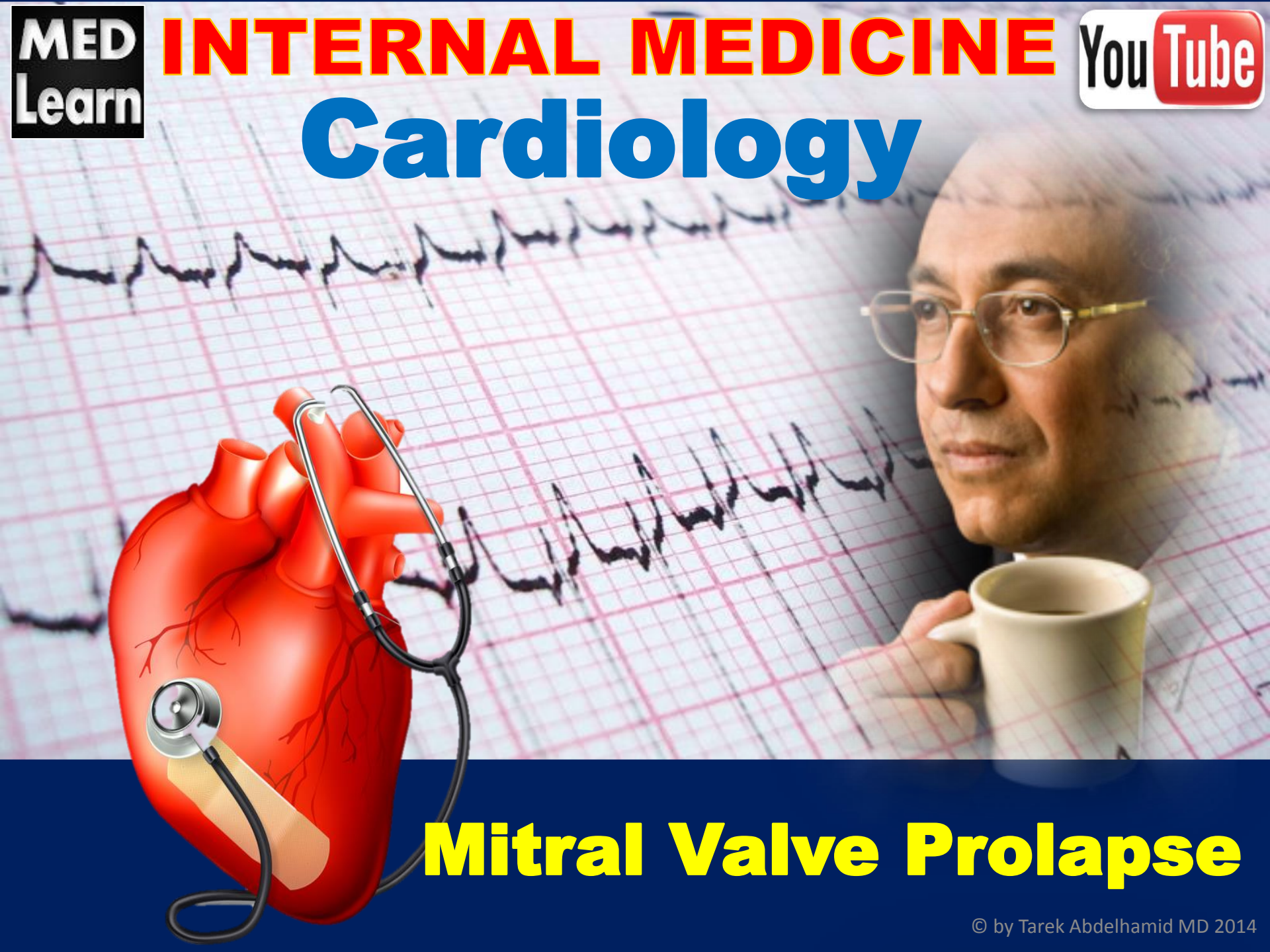
Special Investigations

- ① Dilatation of left ventricle on Echo
- ② No thickened pericardium (or calcification) by CT or MRI
- ③ No equalization of EDP OF LV and RV (**Unlike Constrictive Pericarditis**)
- ④ **Rectal or gingival or Abdominal fat Biopsy** to diagnose amyloidosis
- ⑤ Slightly depressed LV functions by Echo (unlike restrictive pericarditis where you may find normal LV functions)

Treatment

- ① No specific treatment is available
- ② Treatment of hemochromatosis by Desferoxamine may improve the condition
- ③ Steroids may be helpful in some cases of Sarcoidosis
- ④ Anticoagulants are given to prevent thromboembolism (which may occur due to stagnation of blood within the dysfunctional heart)

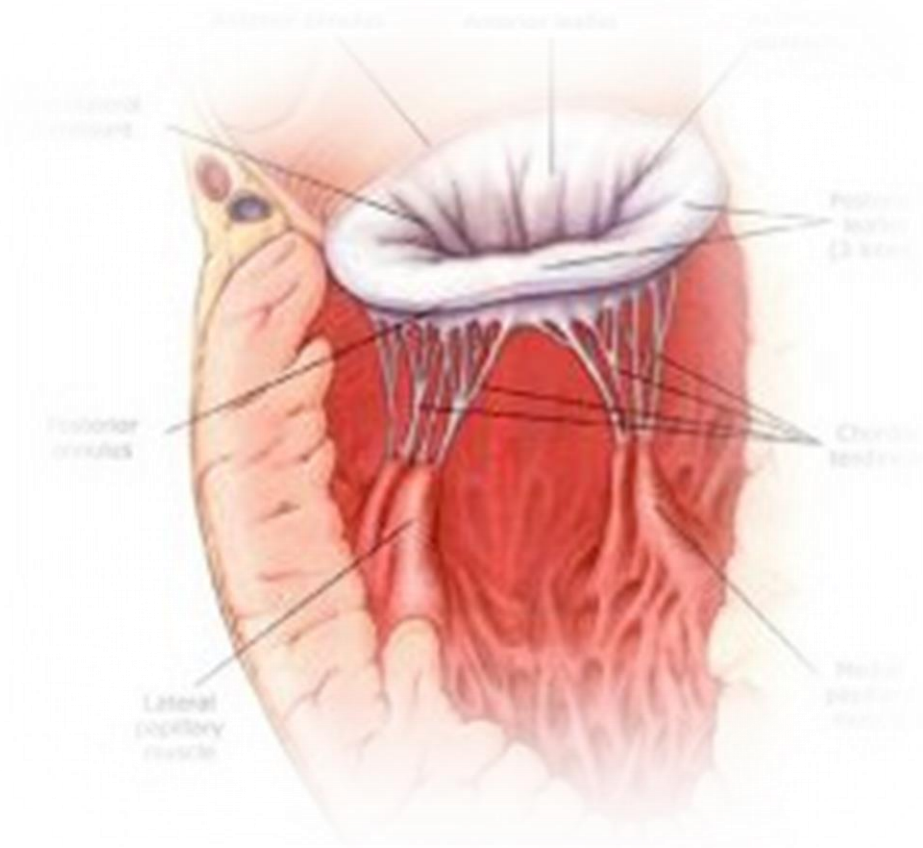
Cardiology



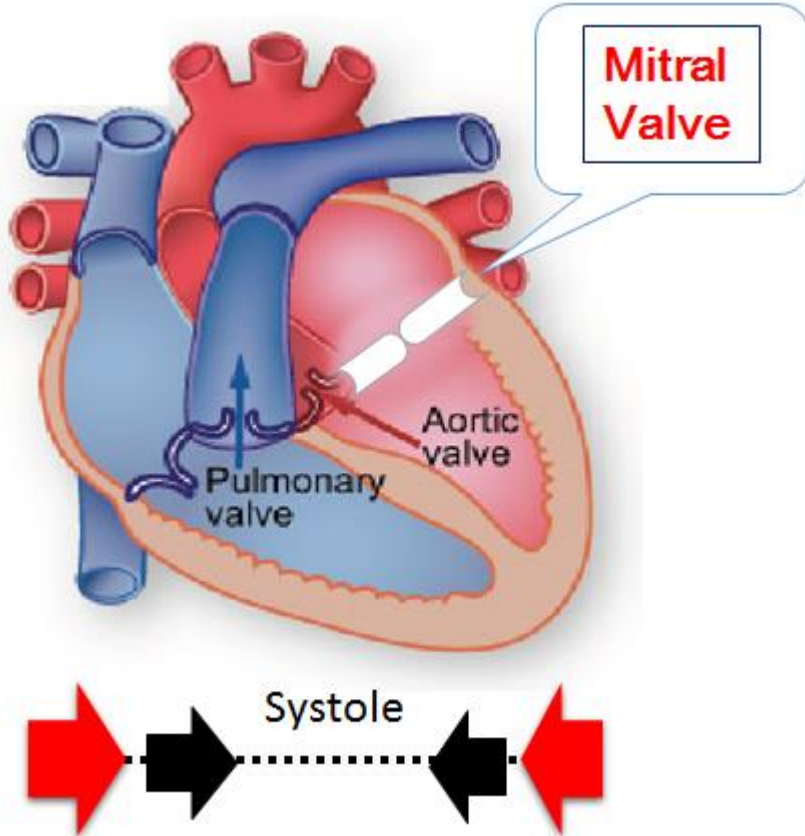
Mitral Valve Prolapse

Definition

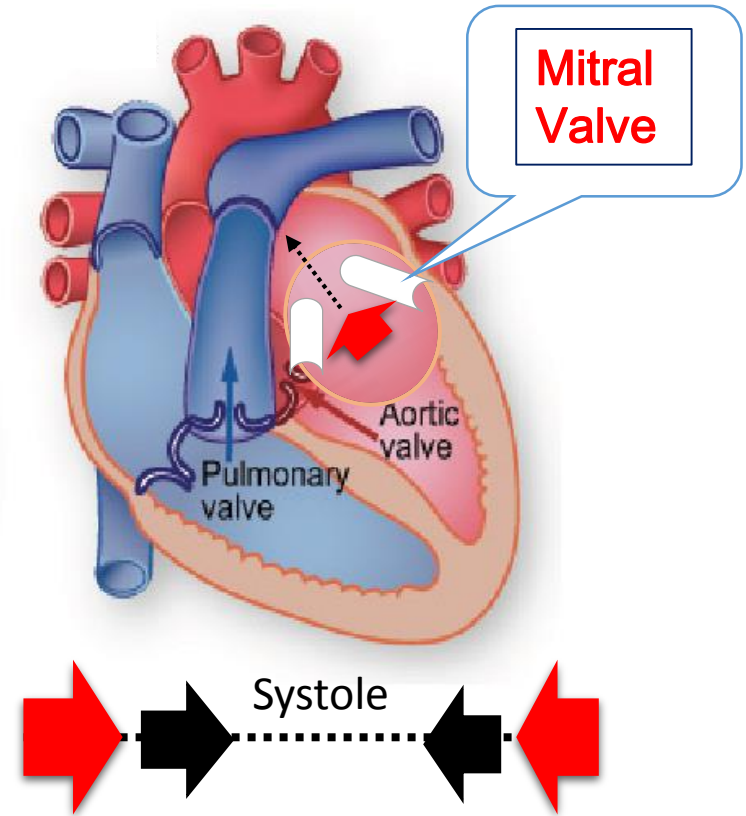
A relatively common but highly variable clinical syndrome caused by an abnormality of the Mitral Valve apparatus that causes its prolapse into the atrium in mild Systole (Mid Systolic Click Syndrome)



Normally

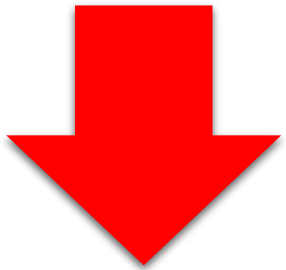


MVP



Aetiology

The exact cause is Unknown- however reduced production of type III Collagen has been incriminated. Another possible mechanism is related to myxomatous degeneration of the papillary muscles.



**Type III
Collagen**

**Myxomatous
degeneration of
Papillary Muscles**

Clinical Picture

Dizziness
or Syncope

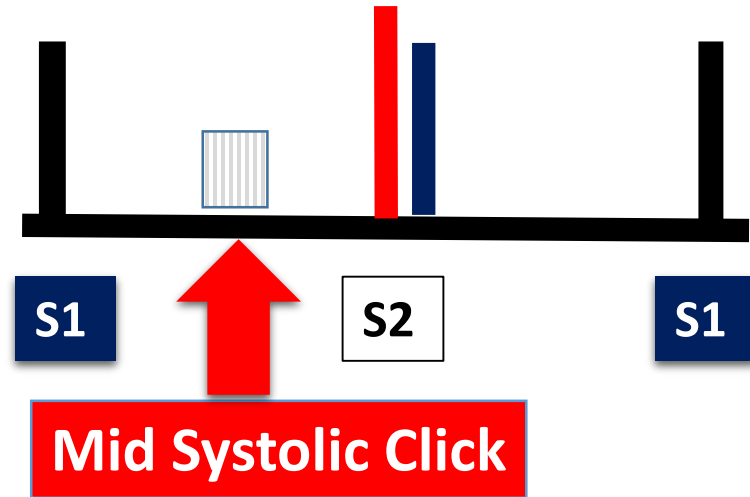
TIA's

Palpitations

Chest Pain

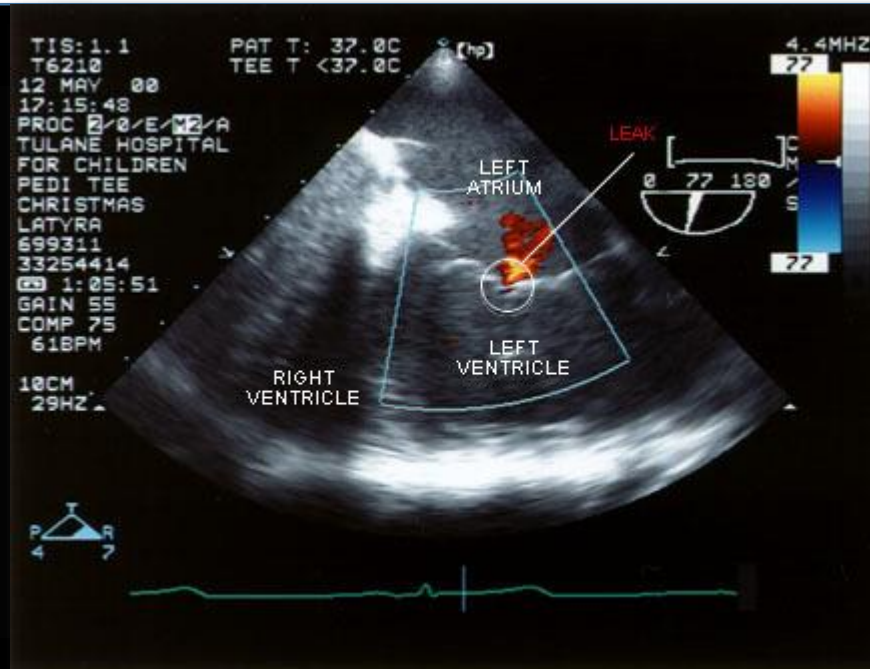
Embolism

SBE



Special Investigations

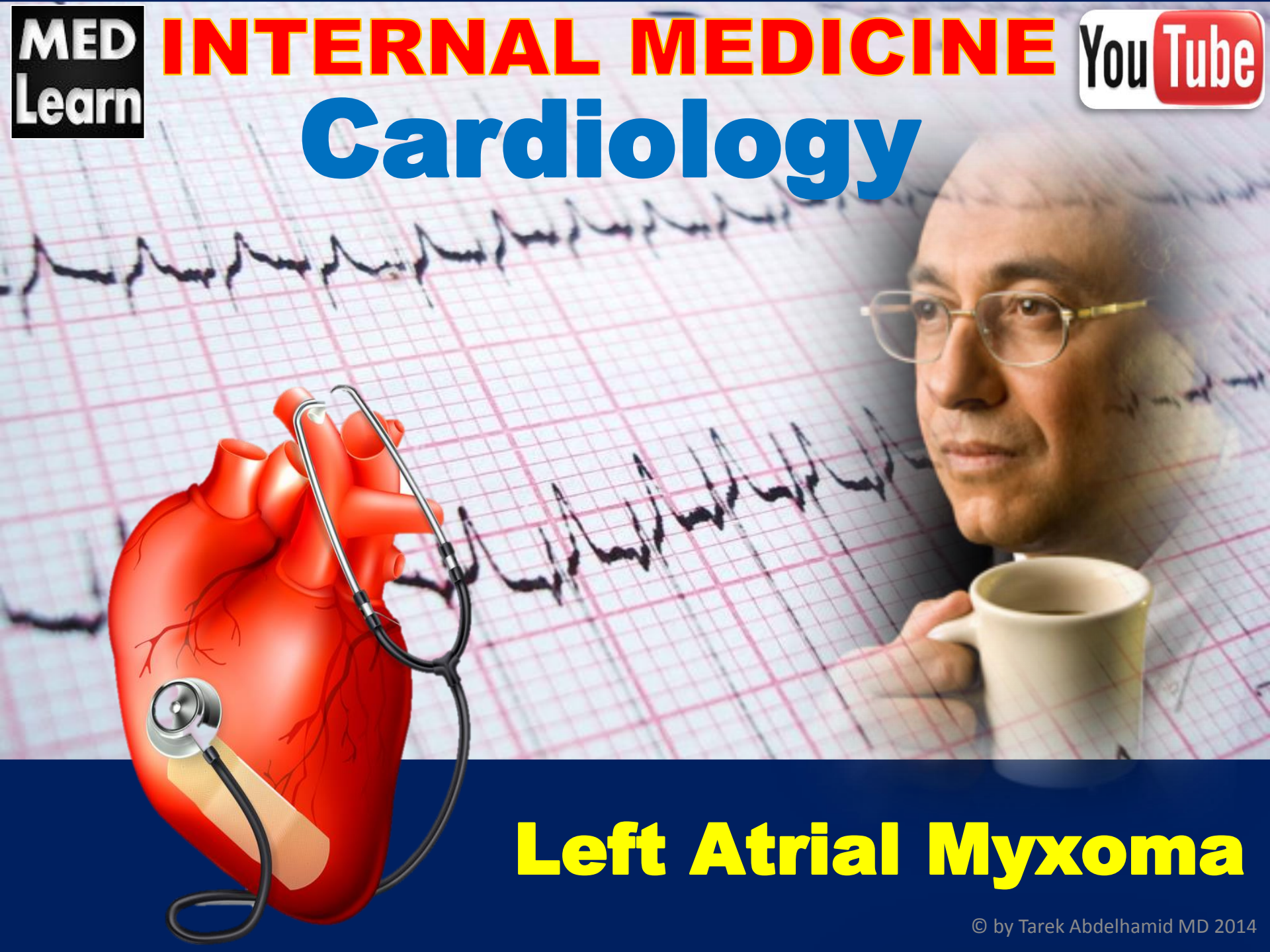
2 Dimensional Echo



Treatment

- ① Beta Blockers (?! Reduce chest pain)
- ② Treatment of associated arrhythmias
- ③ Prophylaxis against SBE

Cardiology

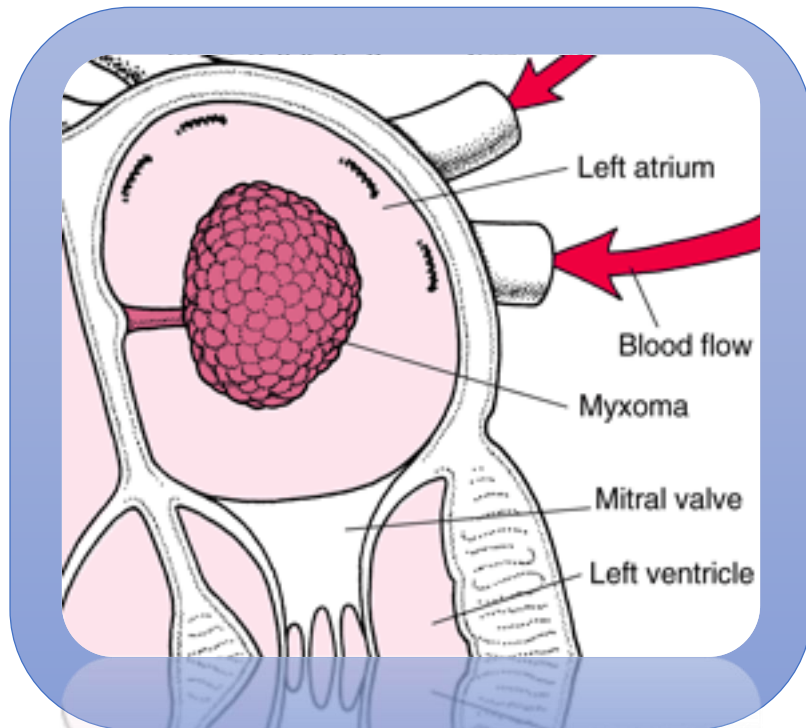


Left Atrial Myxoma

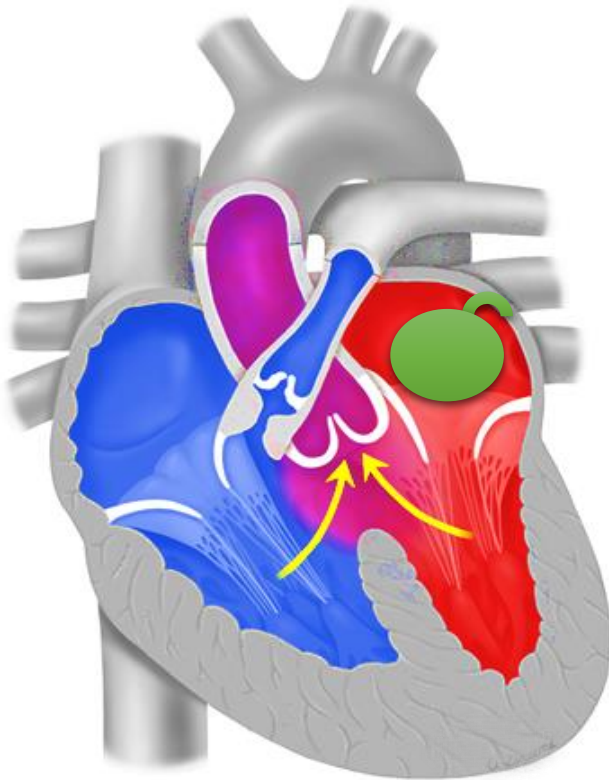
Definition



A benign tumor that originates in the left atrium causing several manifestations.



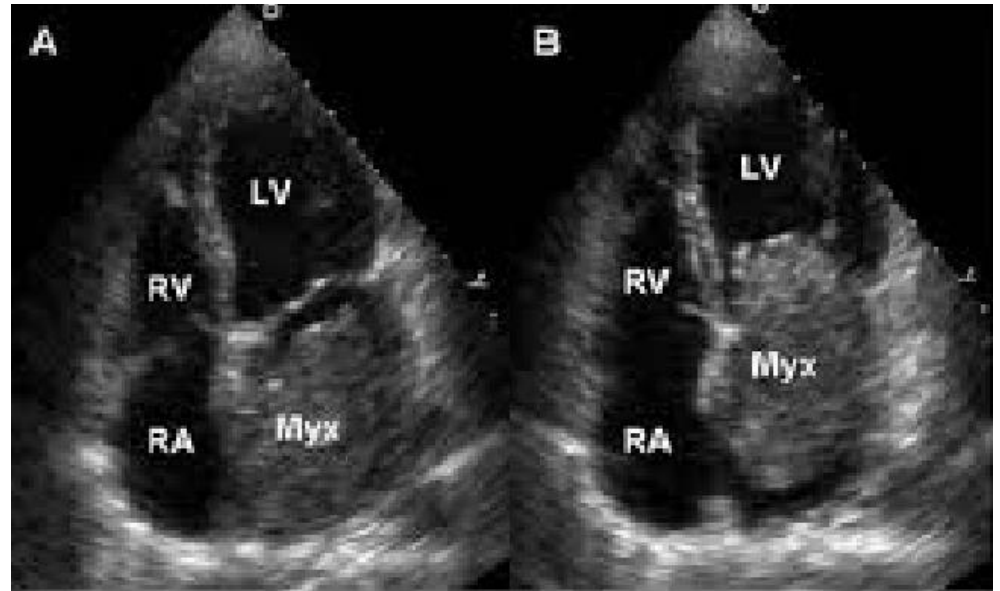
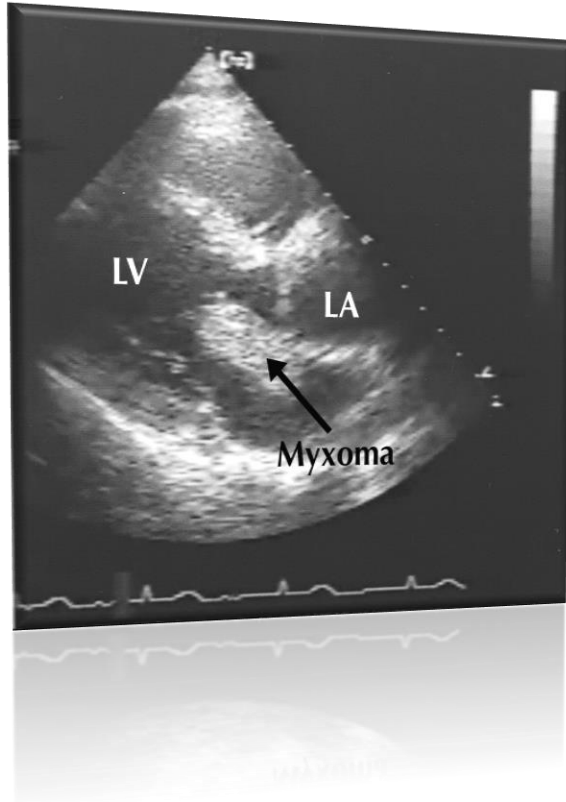
Positional change of Symptoms is Characteristic



- ➡ ① May Obstruct the Mitral Valve causing features of Mitral Stenosis e.g. MDM- Lung Congestion- LCO symptoms
- ➡ ② May Detach causing features of systemic embolization e.g. Stroke
- ➡ ③ May Degenerate causing release of protein antigens that triggers a systemic inflammatory reaction e.g. fever - Malaise - weight loss- Increase ESR- and Leukocytosis

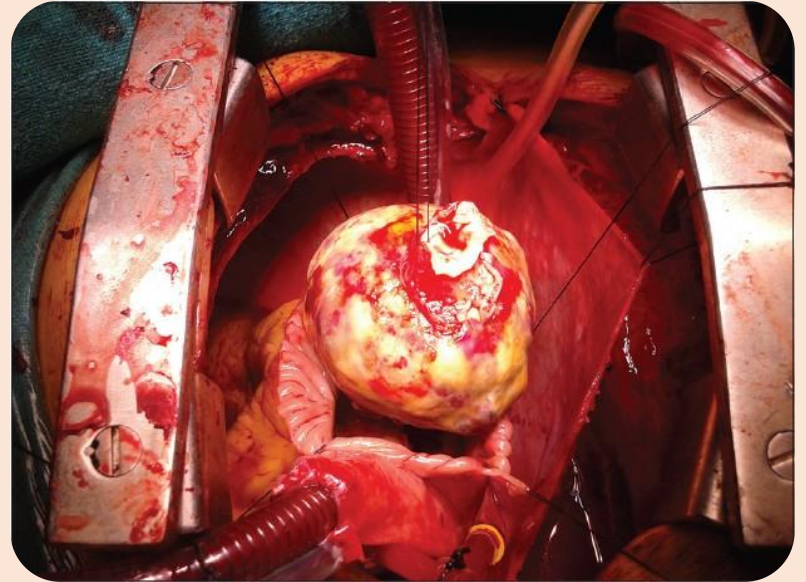
Special Investigations

Echo is diagnostic

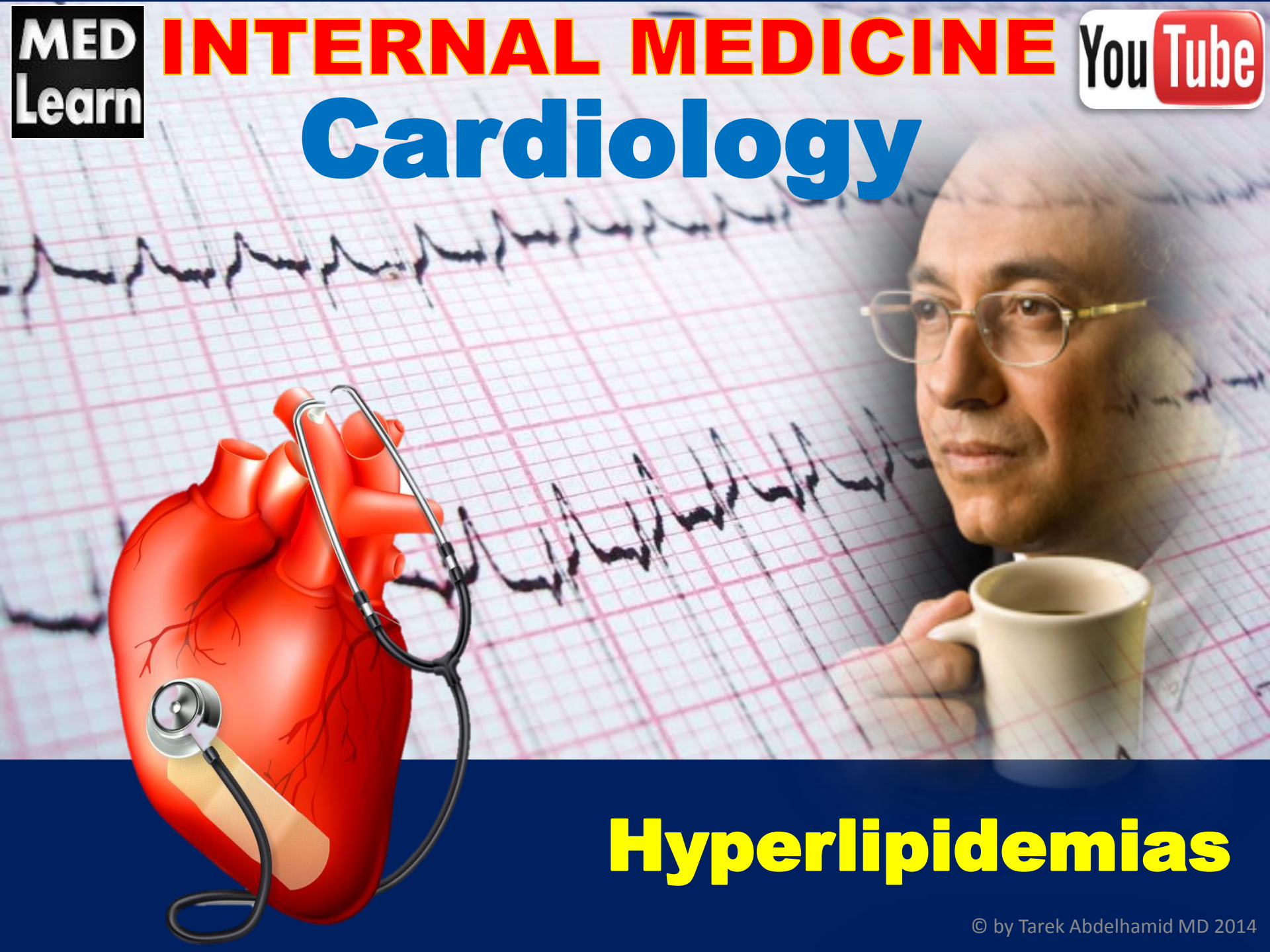


Treatment

**Surgical
treatment
is curative**



Cardiology



Hyperlipidemias

Causes of Hyperlipidemia



Primary



Secondary



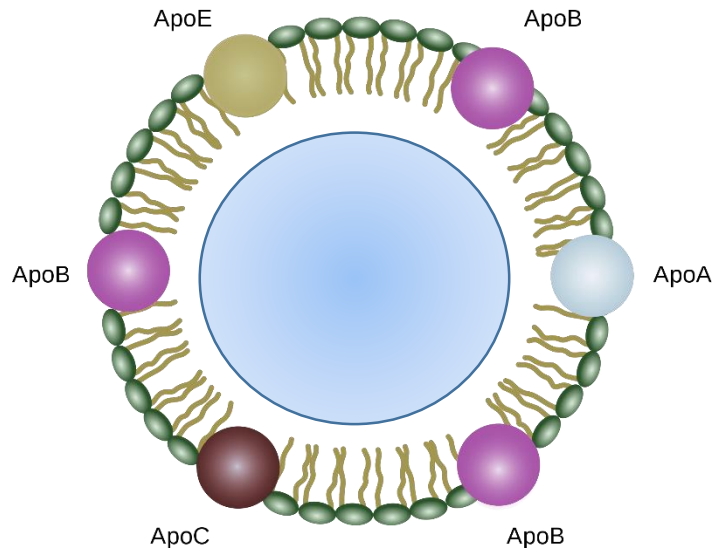
Alcohol
Basement Membrane affection in Nephrotic Syndrome
Cigarette smoking
Diabetes Mellitus (Type II)
Drugs e.g. Anticonvulsants-Beta Blockers-Thiazide Diuretics
Endocrinal: Hypothyroidism - Estrogen (Contraceptive Pills)
Extra and Intrahepatic biliary obstruction
Fatty (Obesity)



Must be excluded before starting therapy by Antihyperlipidemic drugs



Types of Lipoproteins



Chylomicrons

IDL

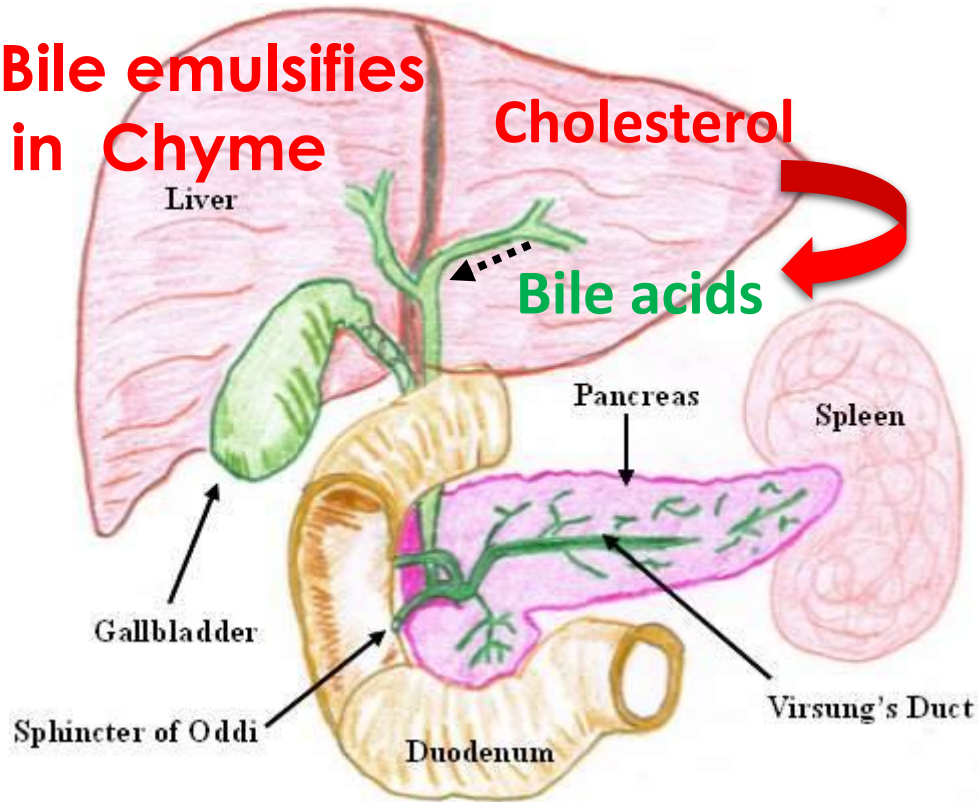
LDL (+++ Cholesterol)

VLDL (+++ TG)

HDL

Step 1

① Bile emulsifies
fat in Chyme



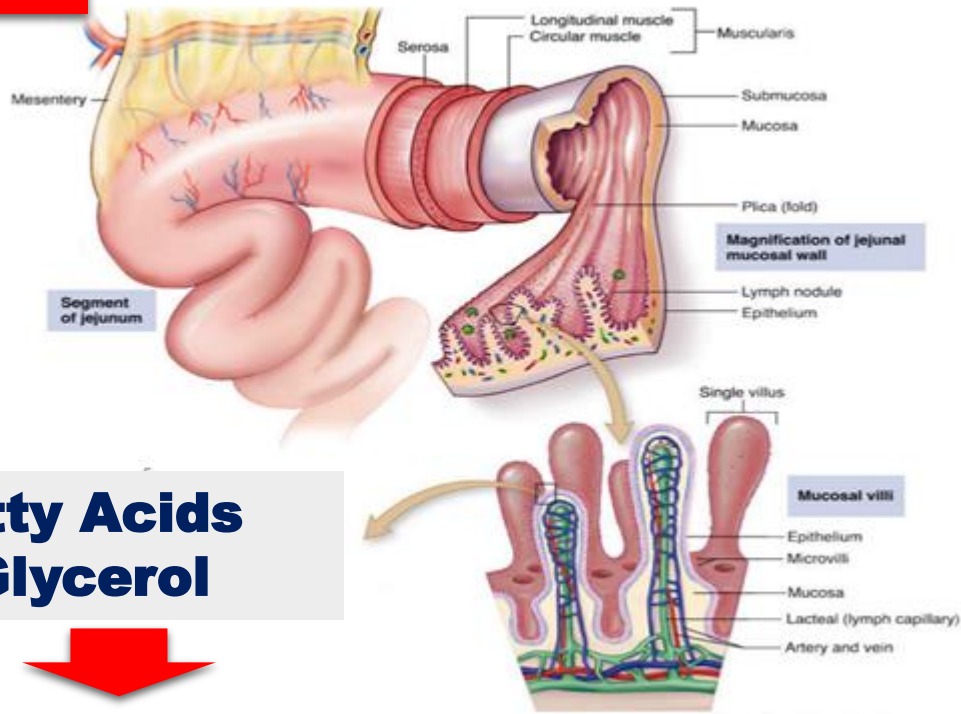
② Pancreatic lipase

Triglycerides



**Fatty Acids
+ Glycerol**

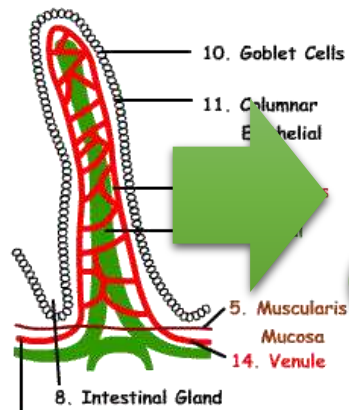
Step 2



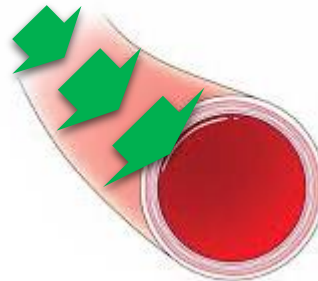
**Fatty Acids
+ Glycerol**



Triglycerides

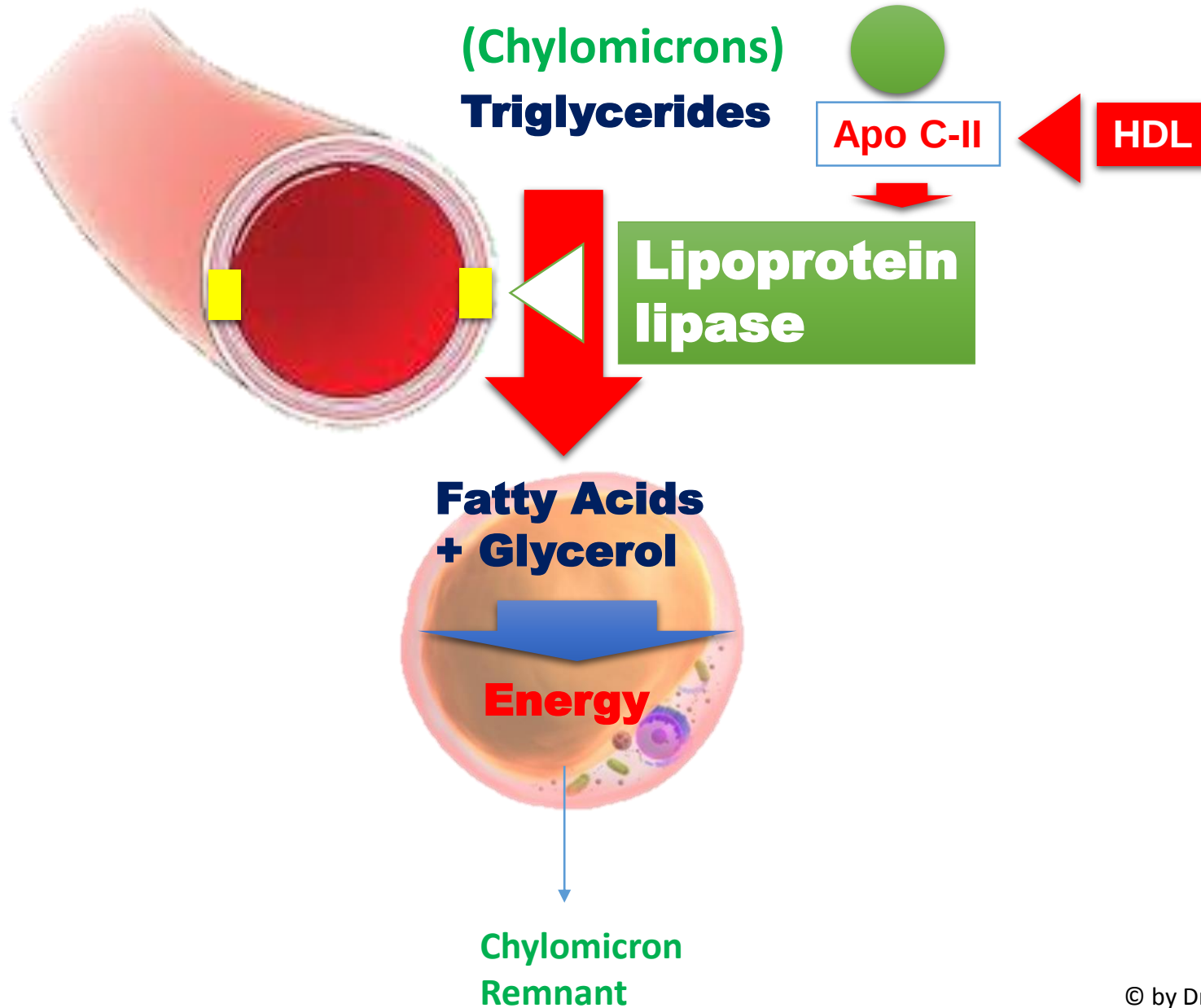


Chylomicrons



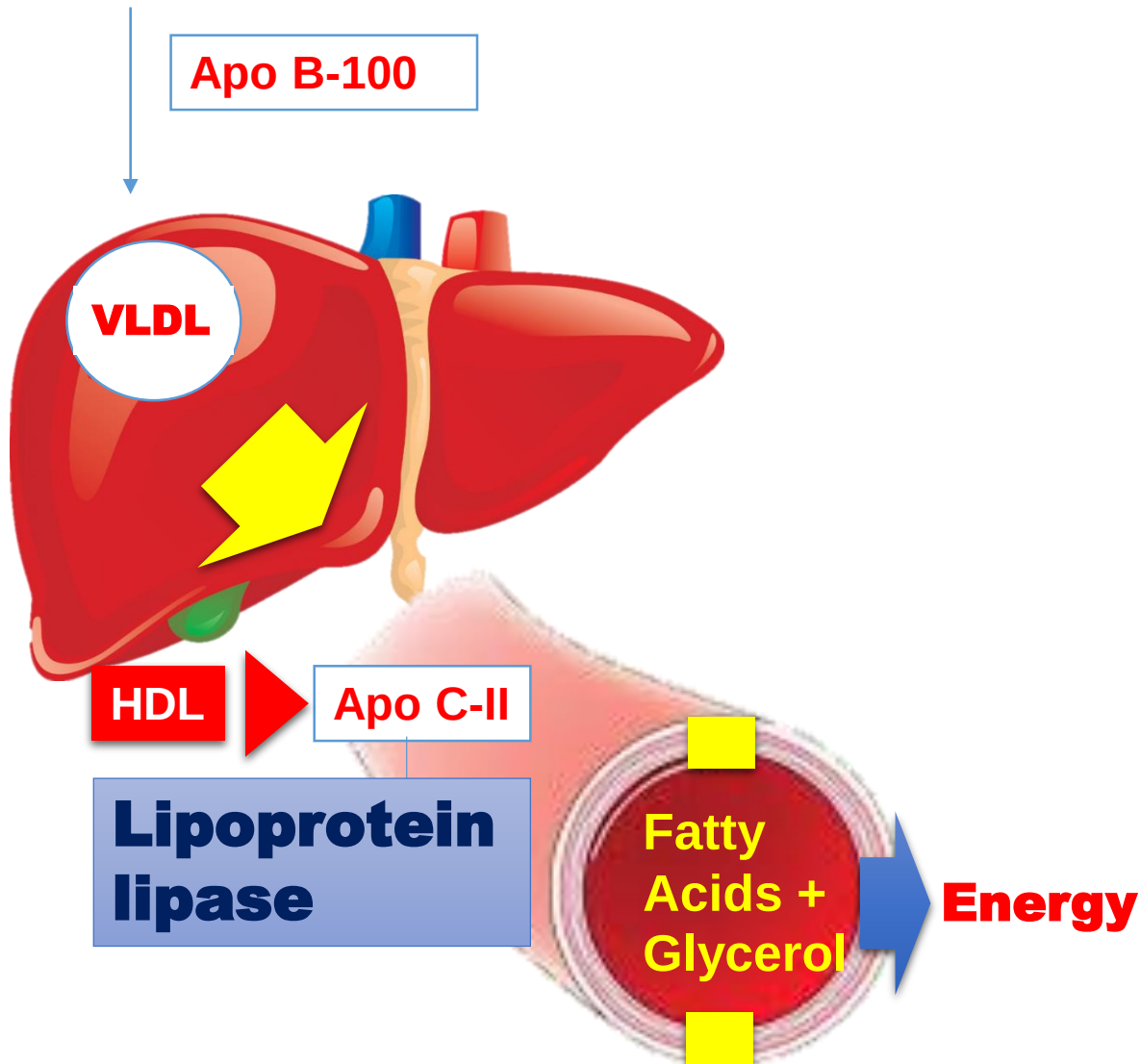
Apo B-48

Step 3



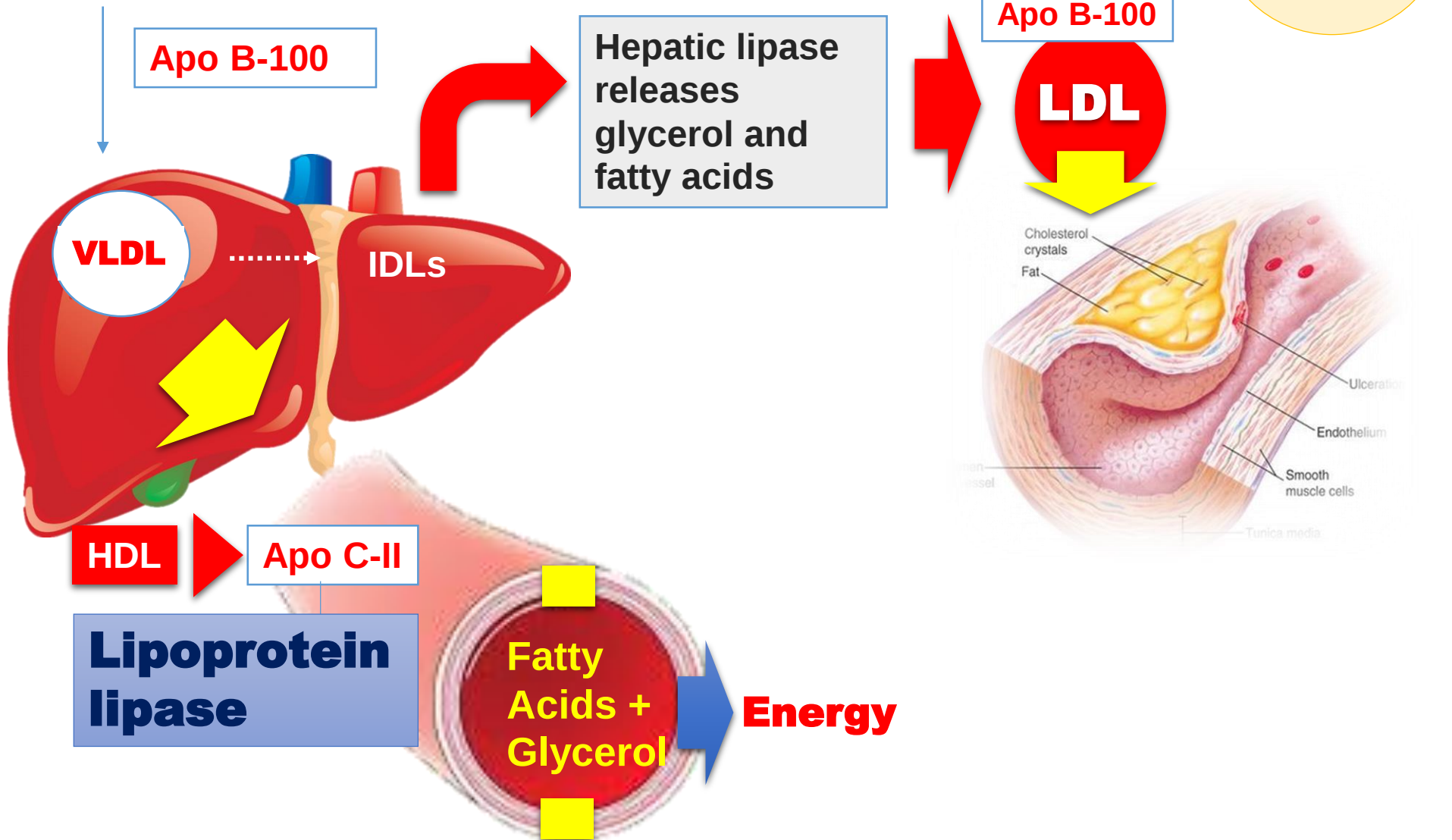
Step 4

*Triglycerides+
cholesteryl esters*



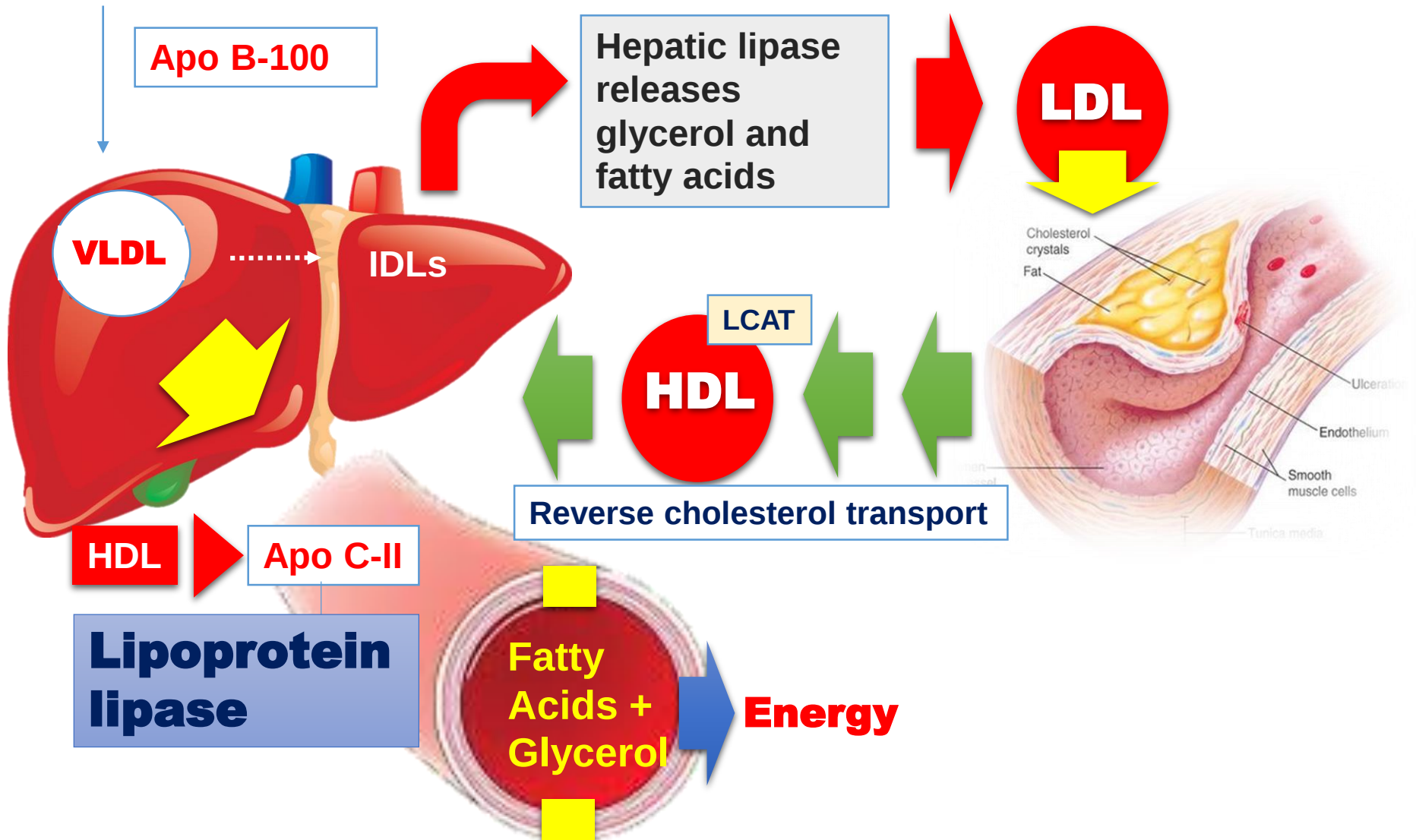
Step 5

*Triglycerides+
cholesteryl esters*



Step 5

*Triglycerides+
cholesteryl esters*





Cholesterol

LDL

① **Familial Hypercholesterolemia(AD)**

Associated with Tendon Xanthoma especially in Tendo Achilles- due to decreased number of LDL Receptors

② **Apo B100 deficiency**

Associated with diminished affinity of LDL Receptors- caused by missense mutation at amino acid number 3500

③ **Polygenic Hypercholesterolemia:**

Multifactorial Inheritance- No tendon Xanthoma





Triglycerides

Chylomicrons

VLDL

① **Familial Hypertriglyceridemia (AD)**

Increase VLDL

② **LPL deficiency**

Associated with Chylomicrons-

Manifests as Pancreatitis- **Eruptive**

Xanthoma- HSM- Pale Retina (Lipemia

Retinalis)- A creamy layer on top of the

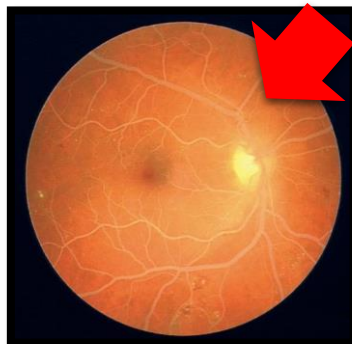
patient plasma (when incubated)- No

increase in LDL activity after heparin)

③ **Polygenic Hypercholesterolemia:**

Multifactorial Inheritance- No tendon

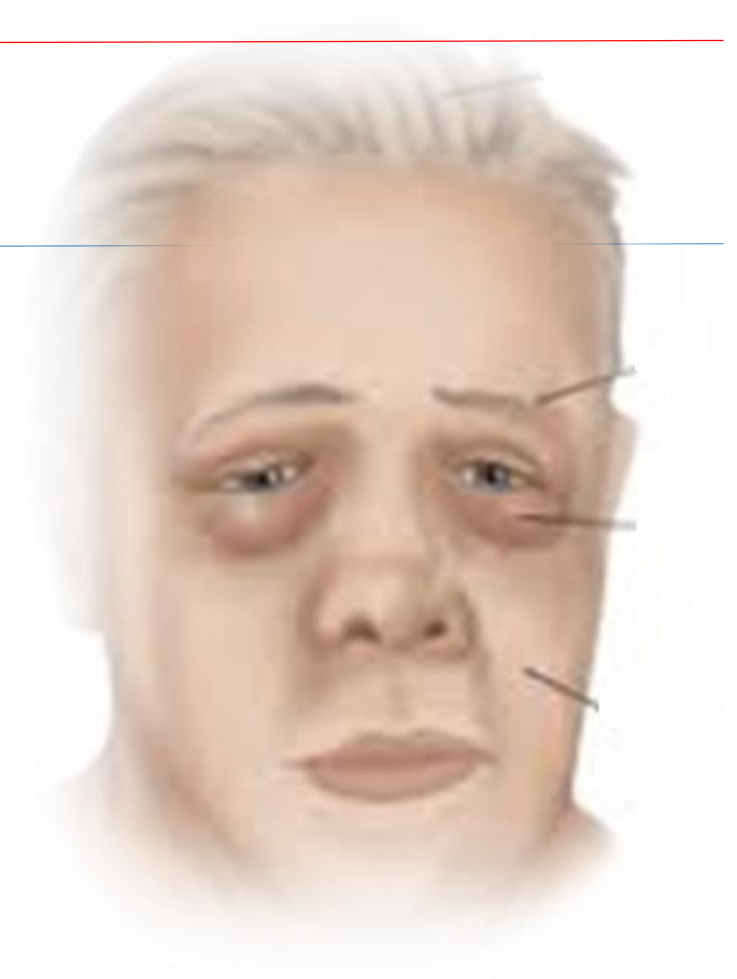
Xanthoma



Treatment

1 Exclude Secondary causes

e.g. Hypothyroidism



Treatment

2 Diet

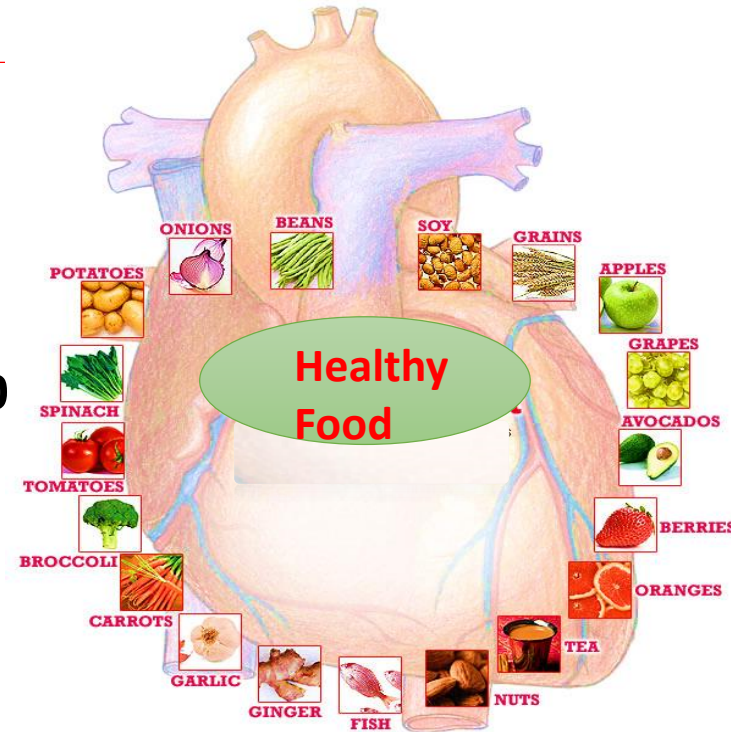
↑ Cholesterol

- 1- Total fat less than 30% of total calories- Saturated fat less than 10% of calories-Cholesterol less than 300 mg per day
- 2- Replace saturated fat with plant sources such as Canola oil- Olive oil- Peanuts- Avocados
- 3- Increase intake of soluble fibers
- 4- Increase intake of Garlic- Soy protein- and Vitamin C (anti oxidant content may decrease cholesterol oxidation)

↑ Triglycerides

- 1- Decrease Alcohol intake
- 2- Decrease simple sugars
- 3- Regular exercise
- 4- Avoid drugs that increase TG e.g. Estrogen
- 5- Increase fish oil intake as it is good source for omega 3

Fatty Acids



Treatment

3 Drugs

Cholesterol

HMG Co A Reductase Inhibitors

Bile acids Binding Resins



Niacin

- 1- Decreases TG**
- 2- Decreases LDL**
- 3- Increases HDL**



Combined Hyperlipidemia

Triglycerides

Fibrates

(Gimfibrizol & Fenfibrate)

HMG Co A Reductase Inhibitors

① Decreases cholesterol biosynthesis

② Increases LDL receptors in liver cells

③ Increase uptake of Cholesterol by the Liver cells

May cause Myopathy

+++ CPK

May decrease liver functions

LFTs

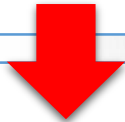
STOP if Liver Enzymes X 3 times

Bile acids Binding Resins

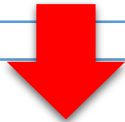
Decreases bile acids absorption in the gut



Increases hepatic bile acid production in the liver



Increases cholesterol uptake by the liver



Decreases Cholesterol Level



Note: May decrease absorption of **Vitamin K**....+++ toxicity of Warfarin...Bleeding



Fibrates

Gimfibrizol & Fenfibrate



Activity of a
hepatic
transcription
factor called PPAR



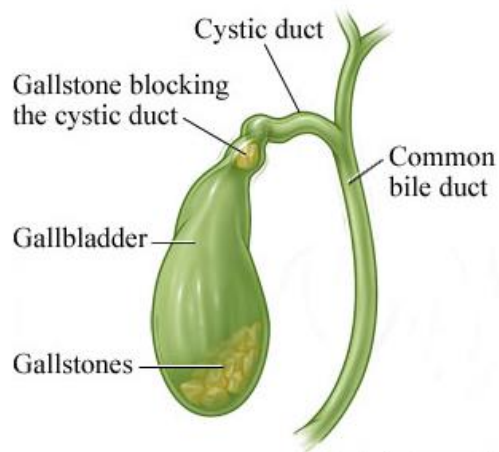
LPL



TG



Incidence of Gall Stones



Side effects:
Abdominal pain and Nausea

Niacin

- ① Very useful in Combined Hyperlipidemia
- ② Increase dose gradually to avoid its side effects
- ③ Can cause PG mediated Headache
(Premedication with Aspirin?!)
- ④ Intolerance is common
- ⑤ May cause Liver Toxicity

